

## WEST Search History





DATE: Wednesday, May 31, 2006

<u>Hide?</u>	<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>
	<i>DB=PGPB,USPT,EPAB; PLUR=YES; OP=ADJ</i>		
<input type="checkbox"/>	L51	L50 not @ay>2002	242
<input type="checkbox"/>	L50	L49 and polymer	342
<input type="checkbox"/>	L49	L48 and L11	375
<input type="checkbox"/>	L48	L45 and L3	520
<input type="checkbox"/>	L47	L45 and L1	3
<input type="checkbox"/>	L46	L45 and L1	0
<input type="checkbox"/>	L45	L44 or L43 or L42	43146
<input type="checkbox"/>	L44	(424/484  424/486  424/489  424/490  424/491)![CCLS]	7810
<input type="checkbox"/>	L43	(514/962  514/963)![CCLS]	773
<input type="checkbox"/>	L42	(435/6)![CCLS]	34779
<input type="checkbox"/>	L41	L40 and L9	18
<input type="checkbox"/>	L40	L36 not L39	18
<input type="checkbox"/>	L39	L36 not L37	112
<input type="checkbox"/>	L38	L37 not L36	337
<input type="checkbox"/>	L37	L29 or L28	355
<input type="checkbox"/>	L36	L35 not @ay>2001	130
<input type="checkbox"/>	L35	L34 and L9	390
<input type="checkbox"/>	L34	L33 and L11	478
<input type="checkbox"/>	L33	L32 and L2	676
<input type="checkbox"/>	L32	L31 and L7	716
<input type="checkbox"/>	L31	L30 or L29 or L28	1570
<input type="checkbox"/>	L30	L27.clm.	1396
<input type="checkbox"/>	L29	L27.ab.	337
<input type="checkbox"/>	L28	L27.ti.	163
<input type="checkbox"/>	L27	L26 or L19	11051
<input type="checkbox"/>	L26	interleukin NEAR2 12	3542
<input type="checkbox"/>	L25	L24 not @ay>2002	16
<input type="checkbox"/>	L24	L23 and L9	34
<input type="checkbox"/>	L23	L22 and L2	37
<input type="checkbox"/>	L22	L21 and L11	40
<input type="checkbox"/>	L21	L20 and L7	58

<input type="checkbox"/>	L20	L19.ab.	258
<input type="checkbox"/>	L19	IL NEAR2 12	9726
<input type="checkbox"/>	L18	L17 not @py>2001	8
<input type="checkbox"/>	L17	L16 and L9	54
<input type="checkbox"/>	L16	L7 and L15	64
<input type="checkbox"/>	L15	L14 and L3	113
<input type="checkbox"/>	L14	L11.ab.	33131
<input type="checkbox"/>	L13	L3 and L6	3453
<input type="checkbox"/>	L12	L10 and L11	2
<input type="checkbox"/>	L11	liposom\$ or microspher\$ or capsula\$	265751
<input type="checkbox"/>	L10	L8 and L9	5
<input type="checkbox"/>	L9	oral\$	207750
<input type="checkbox"/>	L8	L7 and L5	5
<input type="checkbox"/>	L7	gastrointestin\$ or esophag\$ or gastic? or intestin\$ or colorectal\$	123970
<input type="checkbox"/>	L6	gastrointestin\$ or esophag\$ or gastic? or intestin? or colorectal?	92112
<input type="checkbox"/>	L5	L2 and L4	8
<input type="checkbox"/>	L4	L3.ab.	25
<input type="checkbox"/>	L3	sulindac	5294
<input type="checkbox"/>	L2	cancer\$ or tumor\$ or neoplas\$	190275
<input type="checkbox"/>	L1	egilmez.in.	7

END OF SEARCH HISTORY

\$%^STN;HighlightOn= \*\*\*;HighlightOff=\*\*\* ;

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Welcome to STN International! Enter x:x

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NEWS 1 Web Page URLs for STN Seminar Schedule - N. America  
NEWS 2 "Ask CAS" for self-help around the clock  
NEWS 3 SEP 09 ACD predicted properties enhanced in REGISTRY/ZREGISTRY  
NEWS 4 OCT 03 MATHDI removed from STN  
NEWS 5 OCT 04 CA/CAPLUS-Canadian Intellectual Property Office (CIPO) added  
to core patent offices  
NEWS 6 OCT 13 New CAS Information Use Policies Effective October 17, 2005  
NEWS 7 OCT 17 STN(R) AnaVist(TM), Version 1.01, allows the export/download  
of CAPLUS documents for use in third-party analysis and  
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NEWS 8 OCT 27 Free KWIC format extended in full-text databases  
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NEWS 12 NOV 30 REGISTRY/ZREGISTRY on STN(R) enhanced with experimental  
spectral property data  
NEWS 13 DEC 05 CASREACT(R) - Over 10 million reactions available  
  
NEWS EXPRESS DECEMBER 02 CURRENT VERSION FOR WINDOWS IS V8.01,  
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),  
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FILE 'HOME' ENTERED AT 09:22:46 ON 14 DEC 2005

=> file reg

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		0.21	0.21

FILE 'REGISTRY' ENTERED AT 09:22:55 ON 14 DEC 2005  
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STRUCTURE FILE UPDATES: 13 DEC 2005 HIGHEST RN 869843-02-7

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH JULY 14, 2005

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\*\*\*\*\*

\*  
\* The CA roles and document type information have been removed from \*  
\* the IDE default display format and the ED field has been added, \*  
\* effective March 20, 2005. A new display format, IDERL, is now \*  
\* available and contains the CA role and document type information. \*  
\*

\*\*\*\*\*

Structure search iteration limits have been increased. See HELP SLIMITS for details.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

<http://www.cas.org/ONLINE/UG/regprops.html>

=> E "SULINDAC"/CN 25

E1	1	SULIKOL K/CN
E2	1	SULIN/CN
E3	1	--> SULINDAC/CN
E4	1	SULINDAC B .OMEGA.-N-METHYL-L-ARGININE SALT/CN
E5	1	SULINDAC B .OMEGA.-N-NITRO-L-ARGININE METHYL ESTER SALT/
E6	1	SULINDAC B .OMEGA.-N-NITRO-L-ARGININE SALT/CN
E7	1	SULINDAC ETHYL ESTER/CN
E8	1	SULINDAC SODIUM/CN
E9	1	SULINDAC SULFIDE/CN
E10	1	SULINDAC SULFONE/CN
E11	1	SULINDAC SULFOXIDE/CN
E12	1	SULINDAC-QUINOLINE/CN
E13	1	SULINEX/CN
E14	1	SULINOL/CN
E15	1	SULIODOVIZOL/CN
E16	1	SULISATIN/CN
E17	1	SULISATIN DISODIUM SALT/CN
E18	1	SULISATIN SODIUM/CN
E19	1	SULISATINE SODIUM/CN
E20	1	SULISOBENZONE/CN
E21	1	SULJEX/CN
E22	1	SULKA/CN
E23	1	SULKA K BOLUSES/CN
E24	1	SULKA N/CN
E25	1	SULKOR/CN

=> S E3

L1 1 SULINDAC/CN

=> file caplus

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		5.03	5.24

FILE 'CAPLUS' ENTERED AT 09:23:34 ON 14 DEC 2005

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FILE COVERS 1907 - 14 Dec 2005 VOL 143 ISS 25  
FILE LAST UPDATED: 13 Dec 2005 (20051213/ED)

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=> s l1

L2 1426 L1

=> s gastrointestinal or esophag? or gastic? or intestin? or colorect?

2 GASTROINTESTINAL

15568 ESOPHAG?

4 GASTIC?

239459 INTESTIN?

18675 COLORECT?

L3 254068 GASTROINTESTINAL OR ESOPHAG? OR GASTIC? OR INTESTIN?  
T?

=> s cancer? or tumor? or neoplas? or polyp?

277857 CANCER?

411659 TUMOR?

431921 NEOPLAS?

438716 POLYP?

L4 1099978 CANCER? OR TUMOR? OR NEOPLAS? OR POLYP?

=> s l4 and l3

L5 65506 L4 AND L3

=> s l5 and l2

L6 234 L5 AND L2

=> s oral?

L7 243958 ORAL?

=> s l7 and l6

L8 30 L7 AND L6

=> s l2 (l) l4

L9 186 L2 (L) L4

=> s l9 and l3

L10 121 L9 AND L3

=> s l10 and l7

L11 14 L10 AND L7

=> s l14 not py>2002

L14 NOT FOUND

The L-number entered could not be found. To see the definition of L-numbers, enter DISPLAY HISTORY at an arrow prompt (=>).

=> s l11 not py>2002

3346380 PY>2002

L12 9 L11 NOT PY>2002

=> d ibib 1-4

L12 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:723268 CAPLUS

DOCUMENT NUMBER: 138:13001

TITLE: A mouse model of human \*\*\*oral\*\*\* -  
\*\*\*esophageal\*\*\* cancer

AUTHOR(S): Opitz, Oliver G.; Harada, Hideki; Suliman, Yasir;  
Rhoades, Ben; Sharpless, Norman E.; Kent, Ralph;  
Kopelovich, Levy; Nakagawa, Hiroshi; Rustgi, Anil K.

CORPORATE SOURCE: Division of Gastroenterology, University of  
Pennsylvania, Philadelphia, PA, 19104-2144, USA

SOURCE: Journal of Clinical Investigation (2002), 110(6),  
761-769  
CODEN: JCINAO; ISSN: 0021-9738  
PUBLISHER: American Society for Clinical Investigation  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:259707 CAPLUS  
DOCUMENT NUMBER: 136:379639  
TITLE: Primary chemoprevention of familial adenomatous  
polyposis with sulindac  
AUTHOR(S): Giardiello, Francis M.; Yang, Vincent W.; Hyland,  
Linda M.; Krush, Anne J.; Petersen, Gloria M.;  
Trimbath, Jill D.; Piantadosi, Steven; Garrett,  
Elizabeth; Geiman, Deborah E.; Hubbard, Walter;  
Offerhaus, Johan A.; Hamilton, Stanley R.  
CORPORATE SOURCE: Dep. Med., Johns Hopkins Univ. Sch. Med., Baltimore,  
MD, USA  
SOURCE: New England Journal of Medicine (2002), 346(14),  
1054-1059  
CODEN: NEJMAG; ISSN: 0028-4793  
PUBLISHER: Massachusetts Medical Society  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:564792 CAPLUS  
DOCUMENT NUMBER: 135:127230  
TITLE: Method for inhibiting a tumor  
INVENTOR(S): Nair, Muraleedharan G.; Bourquin, Leslie D.; Seeram,  
Navindra P.; Kang, Soo-Young  
PATENT ASSIGNEE(S): Michigan State University, USA  
SOURCE: PCT Int. Appl., 27 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 2  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001054516	A1	20010802	WO 2001-US1196	20010112
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG CA 2398389 AA 20010802 CA 2001-2398389 20010112 PRIORITY APPLN. INFO.: US 2000-494077 A 20000128 WO 2001-US1196 W 20010112				
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT				

L12 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:476884 CAPLUS  
DOCUMENT NUMBER: 135:282815  
TITLE: Sulindac in familial adenomatous polyposis: Evaluation  
by nuclear morphometry  
AUTHOR(S): Fernandez-Lopez, F.; Conde-Freire, R.; Cadarso-Suarez,  
C.; Garcia-Iglesias, J.; Puente-Dominguez, J. L.;  
Potel-Lesquereux, J.  
CORPORATE SOURCE: General Surgery Department, Hospital Clinico  
Universitario, Santiago de Compostela, Spain  
SOURCE: European Journal of Surgery (2001), 167(5), 375-381  
CODEN: EUJSEH; ISSN: 1102-4151

PUBLISHER: Taylor & Francis Ltd.  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib 5-9

L12 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2000:260877 CAPLUS

DOCUMENT NUMBER: 133:217169

TITLE: Sulindac and acetylsalicylic acid (ASA) - clinical  
relevance in familial adenomatous polyposis

AUTHOR(S): Winde, G.

CORPORATE SOURCE: Klinik und Poliklinik für Allgemeine Chirurgie der  
WWU, Münster, D-48129, Germany

SOURCE: Falk Symposium (1999), 109(Colorectal Cancer), 235-255

CODEN: FASYDI; ISSN: 0161-5580

PUBLISHER: Kluwer Academic Publishers

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

REFERENCE COUNT: 91 THERE ARE 91 CITED REFERENCES AVAILABLE  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2000:147314 CAPLUS

DOCUMENT NUMBER: 132:273995

TITLE: Inhibition of rat colon tumors by sulindac and  
sulindac sulfone is independent of K-ras (codon 12)  
mutation

AUTHOR(S): De Jong, Tanya A.; Skinner, Stewart A.;  
Malcontenti-Wilson, Cathy; Vogliagis, Daphne; Bailey,  
Michael; Van Driel, Ian R.; O'Brien, Paul E.

CORPORATE SOURCE: Department of Surgery, Monash University Medical  
School, Melbourne, 3181, Australia

SOURCE: American Journal of Physiology (2000), 278(2, Pt. 1),  
G266-G272

CODEN: AJPHAP; ISSN: 0002-9513

PUBLISHER: American Physiological Society

DOCUMENT TYPE: Journal

LANGUAGE: English

REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2000:18902 CAPLUS

DOCUMENT NUMBER: 132:44655

TITLE: Rectal epithelial apoptosis in familial adenomatous  
polyposis patients treated with sulindac

AUTHOR(S): Keller, J. J.; Offerhaus, G. J. A.; Polak, M.;  
Goodman, S. N.; Zahurak, M. L.; Hylind, L. M.;  
Hamilton, S. R.; Giardiello, F. M.

CORPORATE SOURCE: Department of Medicine, The Johns Hopkins University  
School of Medicine, Baltimore, MD, 21205, USA

SOURCE: Gut (1999), 45(6), 822-828

CODEN: GUTTAK; ISSN: 0017-5749

PUBLISHER: BMJ Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

REFERENCE COUNT: 57 THERE ARE 57 CITED REFERENCES AVAILABLE  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1996:277228 CAPLUS

DOCUMENT NUMBER: 124:331957

TITLE: Sulindac induced regression of \*\*\*colorectal\*\*\*  
adenomas in familial adenomatous polyposis: Evaluation  
of predictive factors

AUTHOR(S): Giardiello, F. M.; Offerhaus, J. A.; Tersmette, A. C.;  
Hylind, L. M.; Krush, A. J.; Brensinger, J. D.;  
Booker, S. V.; Hamilton, S. R.

CORPORATE SOURCE: School Medicine, Johns Hopkins University, Baltimore,  
MD, 21287, USA

SOURCE: Gut (1996), 38(4), 578-581  
CODEN: GUTTAK; ISSN: 0017-5749  
PUBLISHER: BMJ Publishing Group  
DOCUMENT TYPE: Journal  
LANGUAGE: English

L12 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1991:529697 CAPLUS

DOCUMENT NUMBER: 115:129697

TITLE: Lung tumorigenicity of NNK given \*\*\*orally\*\*\* to  
A/J mice: its application to chemopreventive efficacy  
studies

AUTHOR(S): Castonguay, Andre; Pepin, Pierrot; Stoner, Gary D.

CORPORATE SOURCE: Sch. Pharm., Laval Univ., Quebec, QC, G1K 7P4, Can.

SOURCE: Experimental Lung Research (1991), 17(2), 485-99

CODEN: EXLRDA; ISSN: 0190-2148

DOCUMENT TYPE: Journal

LANGUAGE: English

=> d abs 9

L12 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

AB The ability of five chemopreventive agents to inhibit 4-

(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK)-induced lung tumors in A/J mice was detd. The carcinogen was administered in the drinking water during 7 wk (at doses of 9.2 to 3.1 mg/mouse). Three chemopreventive agents: (dose, g/kg diet) ellagic acid (4.0), 2(3)-BHA (5.0), and sulindac (0.13) inhibited the multiplicity of lung adenomas by 52, 88, and 52%, resp., when compared to NNK controls. .beta.-Carotene + retinol (2.14 + 0.009), in combination, and selenium (0.0022) were ineffective. NNK was absorbed more rapidly from the duodenum than from the stomach and was metabolized in both tissues. The activation of NNK by .alpha.-carbon hydroxylation and its deactivation by pyridine N-oxidn. was more extensive in the duodenum than in the stomach. Carbonyl redn. of NNK was 10 times higher in the duodenum. Liver microsomes were more active than lung microsomes in the .alpha.-carbon hydroxylation of NNK, suggesting that some liver isoenzymes of cytochrome P 450 have a high affinity for NNK. Pyridine N-oxidn. was five times more extensive in lung microsomes than in liver microsomes. Collectively, these results demonstrate that NNK given \*\*\*orally\*\*\* to A/J mice provides a suitable model from which to assess the relative activity and mechanisms of action of chemopreventive agents in pulmonary carcinogenesis.

=> d kwic 9

L12 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

TI Lung tumorigenicity of NNK given \*\*\*orally\*\*\* to A/J mice: its  
application to chemopreventive efficacy studies

AB . . . N-oxidn. was five times more extensive in lung microsomes than in liver microsomes. Collectively, these results demonstrate that NNK given \*\*\*orally\*\*\* to A/J mice provides a suitable model from which to assess the relative activity and mechanisms of action of chemopreventive. . .

IT \*\*\*Intestine\*\*\*, metabolism

(duodenum, (methylnitrosamino)(pyridyl)butanone metab. by,  
chemopreventive agents against lung neoplasm effect on)

IT 68-26-8, Retinol 476-66-4, Ellagic acid 7235-40-7, .beta.-Carotene  
14124-67-5, Selenite 25013-16-5 \*\*\*38194-50-2\*\*\*, Sulindac

RL: BIOL (Biological study)

((methylnitrosamino)(pyridyl)butanone-induced lung \*\*\*neoplasm\*\*\*  
response to)

=> d ibib abs keic 8

'KEIC' IS NOT A VALID FORMAT FOR FILE 'CAPLUS'

The following are valid formats:

ABS ----- GI and AB

ALL ----- BIB, AB, IND, RE

APPS ----- AI, PRAI

BIB ----- AN, plus Bibliographic Data and PI table (default)

CAN ----- List of CA abstract numbers without answer numbers



CBIB ----- AN, plus Compressed Bibliographic Data  
 DALL ----- ALL, delimited (end of each field identified)  
 DMAX ----- MAX, delimited for post-processing  
 FAM ----- AN, PI and PRAI in table, plus Patent Family data  
 FBIB ----- AN, BIB, plus Patent FAM  
 IND ----- Indexing data  
 IPC ----- International Patent Classifications  
 MAX ----- ALL, plus Patent FAM, RE  
 PATS ----- PI, SO  
 SAM ----- CC, SX, TI, ST, IT  
 SCAN ----- CC, SX, TI, ST, IT (random display, no answer numbers;  
                   SCAN must be entered on the same line as the DISPLAY,  
                   e.g., D SCAN or DISPLAY SCAN)  
 STD ----- BIB, IPC, and NCL  
  
 IABS ----- ABS, indented with text labels  
 IALL ----- ALL, indented with text labels  
 IBIB ----- BIB, indented with text labels  
 IMAX ----- MAX, indented with text labels  
 ISTD ----- STD, indented with text labels  
  
 OBIB ----- AN, plus Bibliographic Data (original)  
 OIBIB ----- OBIB, indented with text labels  
  
 SBIB ----- BIB, no citations  
 SIBIB ----- IBIB, no citations  
  
 HIT ----- Fields containing hit terms  
 HITIND ----- IC, ICA, ICI, NCL, CC and index field (ST and IT)  
                   containing hit terms  
 HITRN ----- HIT RN and its text modification  
 HITSTR ----- HIT RN, its text modification, its CA index name, and  
                   its structure diagram  
 HITSEQ ----- HIT RN, its text modification, its CA index name, its  
                   structure diagram, plus NTE and SEQ fields  
 FHITSTR ----- First HIT RN, its text modification, its CA index name, and  
                   its structure diagram  
 FHITSEQ ----- First HIT RN, its text modification, its CA index name, its  
                   structure diagram, plus NTE and SEQ fields  
 KWIC ----- Hit term plus 20 words on either side  
 OCC ----- Number of occurrence of hit term and field in which it occurs

To display a particular field or fields, enter the display field codes. For a list of the display field codes, enter HELP DFIELDs at an arrow prompt (=>). Examples of formats include: TI; TI,AU; BIB,ST; TI,IND; TI,SO. You may specify the format fields in any order and the information will be displayed in the same order as the format specification.

All of the formats (except for SAM, SCAN, HIT, HITIND, HITRN, HITSTR, FHITSTR, HITSEQ, FHITSEQ, KWIC, and OCC) may be used with DISPLAY ACC to view a specified Accession Number.  
 ENTER DISPLAY FORMAT (BIB):end

=> d ibib abs kwic 8

L12 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN  
 ACCESSION NUMBER: 1996:277228 CAPLUS  
 DOCUMENT NUMBER: 124:331957  
 TITLE: Sulindac induced regression of \*\*\*colorectal\*\*\*  
           adenomas in familial adenomatous polyposis: Evaluation  
           of predictive factors  
 AUTHOR(S): Giardiello, F. M.; Offerhaus, J. A.; Tersmette, A. C.;  
               Hylind, L. M.; Krush, A. J.; Brensinger, J. D.;  
               Booker, S. V.; Hamilton, S. R.  
 CORPORATE SOURCE: School Medicine, Johns Hopkins University, Baltimore,  
                       MD, 21287, USA  
 SOURCE: Gut (1996), 38(4), 578-581  
           CODEN: GUTTAK; ISSN: 0017-5749  
 PUBLISHER: BMJ Publishing Group  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English  
 AB Background-Sulindac, a non-steroidal anti-inflammatory drug, causes  
       regression of \*\*\*colorectal\*\*\* adenomas in patients with familial

adenomatous polyposis (FAP) but the response is variable. Specific clin. factors predictive of sulindac induced regression have not been studied. Methods-22 patients with FAP were given sulindac 150 mg \*\*\*orally\*\*\* twice a day. Polyp no. and size were detd. before treatment and at three months. The relation of nine clin. factors to polyp regression (per cent of baseline polyp no. after treatment) was evaluated by univariate and multivariate anal. Results-After three months of sulindac, polyp no. had decreased to 45 per cent of baseline and polyp size to 50 per cent of baseline (p<0.001 and p<0.01, resp.). Univariate anal. showed greater polyp regression in older patients (p=0.004), those with previous colectomy and ileorectal anastomosis (p=0.001), and patients without identifiable mutation of the APC gene responsible for FAP (p=0.05). With multivariate regression anal., response to sulindac treatment was assocd. with previous subtotal colectomy. Conclusions-Sulindac treatment seems effective in producing regression of \*\*\*colorectal\*\*\* adenomas of FAP patients with previous subtotal colectomy regardless of baseline polyp no. and size. Changed sulindac metab., reduced area of the target mucosa, or changed epithelial characteristics after ileorectal anastomosis may explain these findings.

TI Sulindac induced regression of \*\*\*colorectal\*\*\* adenomas in familial adenomatous polyposis: Evaluation of predictive factors

AB Background-Sulindac, a non-steroidal anti-inflammatory drug, causes regression of \*\*\*colorectal\*\*\* adenomas in patients with familial adenomatous polyposis (FAP) but the response is variable. Specific clin. factors predictive of sulindac induced regression have not been studied. Methods-22 patients with FAP were given sulindac 150 mg \*\*\*orally\*\*\* twice a day. Polyp no. and size were detd. before treatment and at three months. The relation of nine clin. factors to polyp regression (per cent of baseline polyp no. after treatment) was evaluated by univariate and multivariate anal. Results-After three months of sulindac, polyp no. had decreased to 45 per cent of baseline and polyp size to 50 per cent of baseline (p<0.001 and p<0.01, resp.). Univariate anal. showed greater polyp regression in older patients (p=0.004), those with previous colectomy and ileorectal anastomosis (p=0.001), and patients without identifiable mutation of the APC gene responsible for FAP (p=0.05). With multivariate regression anal., response to sulindac treatment was assocd. with previous subtotal colectomy. Conclusions-Sulindac treatment seems effective in producing regression of \*\*\*colorectal\*\*\* adenomas of FAP patients with previous subtotal colectomy regardless of baseline polyp no. and size. Changed sulindac metab., reduced area of the target mucosa, or changed epithelial characteristics after ileorectal anastomosis may explain these findings.

ST sulindac \*\*\*colorectal\*\*\* adenomas adenomatous polyposis

IT Neoplasm inhibitors  
(large \*\*\*intestine\*\*\* , sulindac induced regression of  
\*\*\*colorectal\*\*\* adenomas in familial adenomatous polyposis in  
humans)

IT \*\*\*Intestine\*\*\* , neoplasm  
(large, inhibitors, sulindac induced regression of \*\*\*colorectal\*\*\*  
adenomas in familial adenomatous polyposis in humans)

IT \*\*\*38194-50-2\*\*\* , Sulindac

RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(sulindac induced regression of \*\*\*colorectal\*\*\* adenomas in  
familial adenomatous \*\*\*polyposis\*\*\* in humans)

=> d ibib abs kwic 2

L12 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:259707 CAPLUS

DOCUMENT NUMBER: 136:379639

TITLE: Primary chemoprevention of familial adenomatous polyposis with sulindac

AUTHOR(S): Giardiello, Francis M.; Yang, Vincent W.; Hyland, Linda M.; Krush, Anne J.; Petersen, Gloria M.; Trimpath, Jill D.; Piantadosi, Steven; Garrett, Elizabeth; Geiman, Deborah E.; Hubbard, Walter; Offerhaus, Johan A.; Hamilton, Stanley R.

CORPORATE SOURCE: Dep. Med., Johns Hopkins Univ. Sch. Med., Baltimore, MD, USA

SOURCE: New England Journal of Medicine (2002), 346(14), 1054-1059

PUBLISHER: Massachusetts Medical Society

DOCUMENT TYPE: Journal

LANGUAGE: English

**AB** Background: Familial adenomatous polyposis is caused by a germ-line mutation in the adenomatous polyposis coli gene and is characterized by the development of hundreds of \*\*\*colorectal\*\*\* adenomas and, eventually, \*\*\*colorectal\*\*\* cancer. Nonsteroidal antiinflammatory drugs can cause regression of adenomas, but whether they can prevent adenomas is unknown. Methods: The authors conducted a randomized, double-blind, placebo-controlled study of 41 young subjects (age range, 8 to 25 yr) who were genotypically affected with familial adenomatous polyposis but phenotypically unaffected. The subjects received either 75 or 150 mg of sulindac \*\*\*orally\*\*\* twice a day or identical-appearing placebo tablets for 48 mo. The no. and size of new adenomas and side effects of therapy were evaluated every four months for four years, and the levels of five major prostaglandins were serially measured in biopsy specimens of normal-appearing \*\*\*colorectal\*\*\* mucosa. Results: After four years of treatment, the av. rate of compliance exceeded 76 % in the sulindac group, and mucosal prostaglandin levels were lower in this group than in the placebo group. During the course of the study, adenomas developed in 9 of 21 subjects (43 %) in the sulindac group and 11 of 20 subjects in the placebo group (55 %) (P = 0.54). There were no significant differences in the mean no. (P = 0.69) or size (P = 0.17) of polyps between the groups. Sulindac did not slow the development of adenomas, according to an evaluation involving linear longitudinal methods. Conclusions: Std. doses of sulindac did not prevent the development of adenomas in subjects with familial adenomatous polyposis.

**REFERENCE COUNT:** 23 THERE ARE 23 CITED REFERENCES AVAILABLE  
**RECORD.** ALL CITATIONS AVAILABLE IN THE RE FORMAT

**AB** Background: Familial adenomatous polyposis is caused by a germ-line mutation in the adenomatous polyposis coli gene and is characterized by the development of hundreds of \*\*\*colorectal\*\*\* adenomas and, eventually, \*\*\*colorectal\*\*\* cancer. Nonsteroidal antiinflammatory drugs can cause regression of adenomas, but whether they can prevent adenomas is unknown. Methods: The authors conducted a randomized, double-blind, placebo-controlled study of 41 young subjects (age range, 8 to 25 yr) who were genotypically affected with familial adenomatous polyposis but phenotypically unaffected. The subjects received either 75 or 150 mg of sulindac \*\*\*orally\*\*\* twice a day or identical-appearing placebo tablets for 48 mo. The no. and size of new adenomas and side effects of therapy were evaluated every four months for four years, and the levels of five major prostaglandins were serially measured in biopsy specimens of normal-appearing \*\*\*colorectal\*\*\* mucosa. Results: After four years of treatment, the av. rate of compliance exceeded 76 % in the sulindac group, and mucosal prostaglandin levels were lower in this group than in the placebo group. During the course of the study, adenomas developed in 9 of 21 subjects (43 %) in the sulindac group and 11 of 20 subjects in the placebo group (55 %) (P = 0.54). There were no significant differences in the mean no. (P = 0.69) or size (P = 0.17) of polyps between the groups. Sulindac did not slow the development of adenomas, according to an evaluation involving linear longitudinal methods. Conclusions: Std. doses of sulindac did not prevent the development of adenomas in subjects with familial adenomatous polyposis.

**IT** Prostaglandins

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
 ( \*\*\*colorectal\*\*\* mucosa prostaglandin levels as measure of  
 sulindac local effect in humans with familial adenomatous polyposis)

**IT** Antitumor agents

( \*\*\*colorectal\*\*\* , adenoma; primary chemoprevention of familial  
 adenomatous polyposis with sulindac in humans)

**IT** \*\*\*Intestine\*\*\* , neoplasm

( \*\*\*colorectal\*\*\* , inhibitors, adenoma; primary chemoprevention of  
 familial adenomatous polyposis with sulindac in humans)

**IT** \*\*\*Intestine\*\*\* , neoplasm

(familial polyposis; primary chemoprevention of familial adenomatous  
 polyposis with sulindac in humans)

**IT** \*\*\*Intestine\*\*\*

(large, mucosa; \*\*\*colorectal\*\*\* mucosa prostaglandin levels as  
 measure of sulindac local effect in humans with familial adenomatous  
 polyposis)

**IT** 363-24-6, Prostaglandin E2 551-11-1, Prostaglandin F2.alpha.

13367-85-6, Prostaglandin B2 41598-07-6, Prostaglandin D2 58962-34-8,  
 6-keto-Prostaglandin F1.alpha.

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
( \*\*\*colorectal\*\*\* mucosa prostaglandin levels as measure of  
sulindac local effect in humans with familial adenomatous polyposis)  
IT \*\*\*38194-50-2\*\*\* , Sulindac  
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological  
activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(primary chemoprevention of familial adenomatous \*\*\*polyposis\*\*\*  
with sulindac in humans)

=> d his

(FILE 'HOME' ENTERED AT 09:22:46 ON 14 DEC 2005)

FILE 'REGISTRY' ENTERED AT 09:22:55 ON 14 DEC 2005  
E "SULINDAC"/CN 25

L1 1 S E3

FILE 'CAPLUS' ENTERED AT 09:23:34 ON 14 DEC 2005

L2 1426 S L1  
L3 254068 S GASTROINTESTINAL OR ESOPHAG? OR GASTIC? OR INTESTII  
L4 1099978 S CANCER? OR TUMOR? OR NEOPLAS? OR POLYP?  
L5 65506 S L4 AND L3  
L6 234 S L5 AND L2  
L7 243958 S ORAL?  
L8 30 S L7 AND L6  
L9 186 S L2 (L) L4  
L10 121 S L9 AND L3  
L11 14 S L10 AND L7  
L12 9 S L11 NOT PY>2002

=> s lipsom? or microspher? or encapsulat? or polymer?

74 LIPSOM?  
27180 MICROSPHER?  
55572 ENCAPSULAT?  
1820552 POLYMER?  
84067 POLYMD  
84067 POLYMD  
(POLYMD)  
31147 POLYMG  
326031 POLYMN  
8505 POLYMNS  
327118 POLYMN  
(POLYMN OR POLYMNS)  
1885881 POLYMER?  
(POLYMER? OR POLYMD OR POLYMG OR POLYMN)  
L13 1945587 LIPSOM? OR MICROSPHER? OR ENCAPSULAT? OR POLYMER?

=> s l13 and l12

L14 0 L13 AND L12

=> s l4 and l2

L15 443 L4 AND L2

=> s l9 and l13

L16 12 L9 AND L13

=> s l16 not py>2002

3346380 PY>2002  
L17 3 L16 NOT PY>2002

=> d ibib 1-3

L17 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:430708 CAPLUS

DOCUMENT NUMBER: 135:236055

TITLE: Rat colorectal tumors treated with a range of  
nonsteroidal anti-inflammatory drugs show altered  
cyclooxygenase-2 and cyclooxygenase-1 splice variant  
mRNA expression levels

AUTHOR(S): Vogiagis, Daphne; Brown, Wendy; Glare, Eric M.;  
O'Brien, Paul E.

CORPORATE SOURCE: Department of Surgery, Monash University Medical  
School, Alfred Hospital, Prahran, 3181, Australia

SOURCE: Carcinogenesis (2001), 22(6), 869-874  
CODEN: CRNGDP; ISSN: 0143-3334  
PUBLISHER: Oxford University Press  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1998:457250 CAPLUS  
DOCUMENT NUMBER: 129:76490  
TITLE: Method for treating a tumor with a chemotherapeutic  
agent and nonemulsified ultrapurified  
\*\*\*polymerized\*\*\* hemoglobin solution  
INVENTOR(S): Teicher, Beverly A.; Rausch, Carl W.; Hopkins, Robert  
E., II  
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, USA; Biopure Corp.  
SOURCE: U.S., 16 pp., Cont.-in-part of U. S. Ser. No. 94,501.  
CODEN: USXXAM  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 3  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5776898	A	19980707	US 1995-477110	19950607
US 5679638	A	19971021	US 1993-94501	19930720
PRIORITY APPLN. INFO.:			US 1991-699769	A2 19910514
			US 1993-94501	A2 19930720

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1997:689536 CAPLUS  
DOCUMENT NUMBER: 127:326520  
TITLE: Method for treating a tumor with a chemotherapeutic  
agent  
INVENTOR(S): Teicher, Beverly A.; Rausch, Carl W.; Hopkins, Robert  
E., II  
PATENT ASSIGNEE(S): Biopure Corporation, USA; Dana Farber Cancer Institute  
SOURCE: U.S., 12 pp., Cont.-in-part of U.S. Ser. No.  
699,769,abandoned.  
CODEN: USXXAM  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 3  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5679638	A	19971021	US 1993-94501	19930720
US 5776898	A	19980707	US 1995-477110	19950607
PRIORITY APPLN. INFO.:			US 1991-699769	B2 19910514
			US 1993-94501	A2 19930720

=> d ibib abs kwic 1

L17 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:430708 CAPLUS  
DOCUMENT NUMBER: 135:236055  
TITLE: Rat colorectal tumors treated with a range of  
nonsteroidal anti-inflammatory drugs show altered  
cyclooxygenase-2 and cyclooxygenase-1 splice variant  
mRNA expression levels  
AUTHOR(S): Vogiagis, Daphne; Brown, Wendy; Glare, Eric M.;  
O'Brien, Paul E.  
CORPORATE SOURCE: Department of Surgery, Monash University Medical  
School, Alfred Hospital, Prahran, 3181, Australia  
SOURCE: Carcinogenesis (2001), 22(6), 869-874  
CODEN: CRNGDP; ISSN: 0143-3334  
PUBLISHER: Oxford University Press  
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Nonsteroidal anti-inflammatory drugs (NSAIDs) reduce tumor mass by increasing tumor cell apoptosis and decreasing cell proliferation. The classically recognized targets for NSAID action are the two isoforms of the cyclooxygenase (COX) gene, which is responsible for prostaglandin prodn. In the rat, the COX-1 gene expresses an alternatively spliced mRNA COX-1 splice variant (SV) which may, at best, code for a truncated COX-1 protein. Previously, it was reported that COX-1SV mRNA is differentially expressed in the ageing stomach. In this study, carcinogen-treated rats were treated for 23 wk with the NSAIDs celecoxib, sulindac or sulindac sulfone, while untreated rats received vehicle alone. The nos. and vols. of tumor per animal were recorded and histol. was performed. The competitive \*\*\*polymerase\*\*\* chain reaction, was used to det. whether COX gene expression was altered in colorectal tumors and in regions of adjacent and distant macroscopically normal intestine, from vehicle- or NSAID-treated rats. In addn., COX-1 and COX-2 were immunolocalized in the same tumor and normal colonic tissue. Tumors from animals treated with vehicle or celecoxib expressed elevated levels of COX-2 mRNA in comparison with the adjacent normal mucosa. In contrast, tumors from sulindac- and sulindac sulfone-treated rats expressed less COX-2 mRNA than tumors from vehicle-treated rats. The expression of COX-1 mRNA remained unchanged in all tissues examd. However, COX-1SV mRNA contents were elevated in colorectal tumors and reduced after NSAID treatment to the values in normal colonic mucosa. The results indicate that the antineoplastic actions of NSAIDs may be attributed to COX-dependent and/or COX-independent mechanisms of action. The presence and differential expression of COX-1SV mRNA was also demonstrated in colon tumors. COX-1SV mRNA represents 2% of the total COX-1 mRNA expressed and its role in colon cancer remains to be established.

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Nonsteroidal anti-inflammatory drugs (NSAIDs) reduce tumor mass by increasing tumor cell apoptosis and decreasing cell proliferation. The classically recognized targets for NSAID action are the two isoforms of the cyclooxygenase (COX) gene, which is responsible for prostaglandin prodn. In the rat, the COX-1 gene expresses an alternatively spliced mRNA COX-1 splice variant (SV) which may, at best, code for a truncated COX-1 protein. Previously, it was reported that COX-1SV mRNA is differentially expressed in the ageing stomach. In this study, carcinogen-treated rats were treated for 23 wk with the NSAIDs celecoxib, sulindac or sulindac sulfone, while untreated rats received vehicle alone. The nos. and vols. of tumor per animal were recorded and histol. was performed. The competitive \*\*\*polymerase\*\*\* chain reaction, was used to det. whether COX gene expression was altered in colorectal tumors and in regions of adjacent and distant macroscopically normal intestine, from vehicle- or NSAID-treated rats. In addn., COX-1 and COX-2 were immunolocalized in the same tumor and normal colonic tissue. Tumors from animals treated with vehicle or celecoxib expressed elevated levels of COX-2 mRNA in comparison with the adjacent normal mucosa. In contrast, tumors from sulindac- and sulindac sulfone-treated rats expressed less COX-2 mRNA than tumors from vehicle-treated rats. The expression of COX-1 mRNA remained unchanged in all tissues examd. However, COX-1SV mRNA contents were elevated in colorectal tumors and reduced after NSAID treatment to the values in normal colonic mucosa. The results indicate that the antineoplastic actions of NSAIDs may be attributed to COX-dependent and/or COX-independent mechanisms of action. The presence and differential expression of COX-1SV mRNA was also demonstrated in colon tumors. COX-1SV mRNA represents 2% of the total COX-1 mRNA expressed and its role in colon cancer remains to be established.

IT \*\*\*38194-50-2\*\*\* , Sulindac 59973-80-7, Sulindac sulfone 169590-42-5, Celecoxib

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)

(colorectal \*\*\*tumors\*\*\* treated with nonsteroidal anti-inflammatory drugs show altered cyclooxygenase-2 and cyclooxygenase-1 splice variant mRNA expression)

=> file medline

COST IN U.S. DOLLARS

ENTRY SINCE FILE TOTAL  
SESSION

FULL ESTIMATED COST

64.77 70.01

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE TOTAL

ENTRY      SESSION  
CA SUBSCRIBER PRICE                      -3.65      -3.65

FILE 'MEDLINE' ENTERED AT 09:35:28 ON 14 DEC 2005

FILE LAST UPDATED: 8 DEC 2005 (20051208/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 will soon be available. For details on the 2005 reload, enter HELP RLOAD at an arrow prompt (=>). See also:

<http://www.nlm.nih.gov/mesh/>  
[http://www.nlm.nih.gov/pubs/techbull/nd04/nd04\\_mesh.html](http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html)  
[http://www.nlm.nih.gov/pubs/techbull/nd05/nd05\\_med\\_data\\_changes.html](http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html)  
[http://www.nlm.nih.gov/pubs/techbull/nd05/nd05\\_2006\\_MeSH.html](http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html)

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s SULINDAC/CN

L18      919 SULINDAC/CN

=> s cancer? or tumor? or neoplas? or polyp?

547932 CANCER?

758323 TUMOR?

1455946 NEOPLAS?

155044 POLYP?

L19    1879233 CANCER? OR TUMOR? OR NEOPLAS? OR POLYP?

=> s gastrointestinal or esophag? or gastic? or intestin? or colorect?

1 GASTROINTESTINAL

101857 ESOPHAG?

50 GASTIC?

293936 INTESTIN?

45036 COLORECT?

L20    428581 GASTROINTESTINAL OR ESOPHAG? OR GASTIC? OR INTESTIN?  
T?

=> s l19 and l20

L21      125328 L19 AND L20

=> s l21 and l18

L22      175 L21 AND L18

=> s liposom? or microspher? or encapsulat? or polymer?

30623 LIPOSOM?

21357 MICROSPHER?

15072 ENCAPSULAT?

351141 POLYMER?

L23    407843 LIPOSOM? OR MICROSPHER? OR ENCAPSULAT? OR POLYMER?

=> s l23 and l22

L24      8 L23 AND L22

=> s l24 not py>2002

1733376 PY>2002

L25      6 L24 NOT PY>2002

=> d ibib 1-3

L25 ANSWER 1 OF 6    MEDLINE on STN

ACCESSION NUMBER: 2002696841    MEDLINE

DOCUMENT NUMBER: PubMed ID: 12458338

TITLE:                Effects of long-term administration of sulindac on APC mRNA  
and apoptosis in colons of rats treated with azoxymethane.

AUTHOR:              Kishimoto Y; Yashima K; Morisawa T; Ohishi T; Marumoto A;  
Sano A; Idobe-Fujii Y; Miura N; Shiota G; Murawaki Y;

Hasegawa J  
CORPORATE SOURCE: Division of Pharmacotherapeutics, Department of  
Pathophysiological and Therapeutic Science, Faculty of  
Medicine, Tottori University, 86 Nishicho, Yonago 683-8503,  
Japan.. ykishimo@grape.med.tottori-u.ac.jp

SOURCE: Journal of cancer research and clinical oncology, (2002  
Nov) 128 (11) 589-95. Electronic Publication: 2002-10-04.  
Journal code: 7902060. ISSN: 0171-5216.

PUB. COUNTRY: Germany: Germany, Federal Republic of

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200301

ENTRY DATE: Entered STN: 20021217

Last Updated on STN: 20030118

Entered Medline: 20030117

L25 ANSWER 2 OF 6 MEDLINE on STN

ACCESSION NUMBER: 2001065648 MEDLINE

DOCUMENT NUMBER: PubMed ID: 11093808

TITLE: Growth-suppressive effect of non-steroidal  
anti-inflammatory drugs on 11 colon- \*\*\*cancer\*\*\* cell  
lines and fluorescence differential display of genes whose  
expression is influenced by sulindac.

AUTHOR: Akashi H; Han H J; Iizaka M; Nakamura Y

CORPORATE SOURCE: Laboratory of Molecular Medicine, Human Genome Center,  
Institute of Medical Science, University of Tokyo, Tokyo,  
Japan.

SOURCE: International journal of cancer. Journal international du  
cancer, (2000 Dec 15) 88 (6) 873-80.

Journal code: 0042124. ISSN: 0020-7136.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200012

ENTRY DATE: Entered STN: 20010322

Last Updated on STN: 20010322

Entered Medline: 20001222

L25 ANSWER 3 OF 6 MEDLINE on STN

ACCESSION NUMBER: 2001064500 MEDLINE

DOCUMENT NUMBER: PubMed ID: 11076880

TITLE: Sulindac and a cyclooxygenase-2 inhibitor, etodolac,  
increase APC mRNA in the colon of rats treated with  
azoxymethane.

AUTHOR: Kishimoto Y; Takata N; Jinnai T; Morisawa T; Shiota G;  
Kawasaki H; Hasegawa J

CORPORATE SOURCE: Department of Clinical Pharmacology, Faculty of Medicine,  
Tottori University, 86 Nishicho, Yonago 683-8503, Japan..  
ykishimo@grape.med.tottori-u.ac.jp

SOURCE: Gut, (2000 Dec) 47 (6) 812-9.

Journal code: 2985108R. ISSN: 0017-5749.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 200012

ENTRY DATE: Entered STN: 20010322

Last Updated on STN: 20010322

Entered Medline: 20001222

=> d ibib 4-6

L25 ANSWER 4 OF 6 MEDLINE on STN

ACCESSION NUMBER: 2000295032 MEDLINE

DOCUMENT NUMBER: PubMed ID: 10833474

TITLE: Par-4, a proapoptotic gene, is regulated by NSAIDs in human  
colon carcinoma cells.

AUTHOR: Zhang Z; DuBois R N

CORPORATE SOURCE: Division of Gastroenterology, Department of Medicine and  
Cell Biology, Vanderbilt University Medical Center,  
Veterans Affairs Medical Center, Nashville, Tennessee, USA.



CONTRACT NUMBER: DK47297 (NIDDK)  
P30 CA68485 (NCI)  
PO CA77839 (NCI)  
SOURCE: Gastroenterology, (2000 Jun) 118 (6) 1012-7.  
Journal code: 0374630. ISSN: 0016-5085.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 200006  
ENTRY DATE: Entered STN: 20000629  
Last Updated on STN: 20021219  
Entered Medline: 20000621

L25 ANSWER 5 OF 6 MEDLINE on STN  
ACCESSION NUMBER: 1999333404 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 10403841  
TITLE: Redistribution of activated caspase-3 to the nucleus during  
butyric acid-induced apoptosis.  
AUTHOR: Mandal M; Adam L; Kumar R  
CORPORATE SOURCE: Cell Growth Regulation Laboratory, University of Texas M.D  
Anderson Cancer Center, Houston, Texas, 77030, USA.  
SOURCE: Biochemical and biophysical research communications, (1999  
Jul 14) 260 (3) 775-80.  
Journal code: 0372516. ISSN: 0006-291X.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199908  
ENTRY DATE: Entered STN: 19990827  
Last Updated on STN: 20020420  
Entered Medline: 19990816

L25 ANSWER 6 OF 6 MEDLINE on STN  
ACCESSION NUMBER: 96334961 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 8707116  
TITLE: Sulindac increases the expression of APC mRNA in malignant  
colonic epithelial cells: an in vitro study.  
AUTHOR: Schnitzler M; Dwight T; Robinson B G  
CORPORATE SOURCE: Molecular Genetics Unit, Kolling Institute of Medical  
Research, Royal North Shore Hospital, St Leonards, NSW,  
Australia.  
SOURCE: Gut, (1996 May) 38 (5) 707-13.  
Journal code: 2985108R. ISSN: 0017-5749.  
PUB. COUNTRY: ENGLAND: United Kingdom  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 199609  
ENTRY DATE: Entered STN: 19960919  
Last Updated on STN: 19970203  
Entered Medline: 19960910

=> d ibib abs kwic 4

L25 ANSWER 4 OF 6 MEDLINE on STN  
ACCESSION NUMBER: 2000295032 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 10833474  
TITLE: Par-4, a proapoptotic gene, is regulated by NSAIDs in human  
colon carcinoma cells.  
AUTHOR: Zhang Z; DuBois R N  
CORPORATE SOURCE: Division of Gastroenterology, Department of Medicine and  
Cell Biology, Vanderbilt University Medical Center,  
Veterans Affairs Medical Center, Nashville, Tennessee, USA.  
CONTRACT NUMBER: DK47297 (NIDDK)  
P30 CA68485 (NCI)  
PO CA77839 (NCI)  
SOURCE: Gastroenterology, (2000 Jun) 118 (6) 1012-7.  
Journal code: 0374630. ISSN: 0016-5085.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 200006  
ENTRY DATE: Entered STN: 20000629  
Last Updated on STN: 20021219  
Entered Medline: 20000621

AB BACKGROUND & AIMS: Many reports indicate that nonsteroidal anti-inflammatory drugs (NSAIDs) have antineoplastic effects, but the precise molecular mechanism(s) responsible are unclear. We evaluated the effect of cyclooxygenase (COX) inhibitors (NSAIDs) on human colon carcinoma cells (HCA-7) and identified several genes that are regulated after treatment with NS-398, a selective COX-2 inhibitor. METHODS: Differential display \*\*\*polymerase\*\*\* chain reaction cloning techniques were used to identify genes regulated by treatment with NSAIDs and selective COX-2 inhibitors. RESULTS: A prostate apoptosis response 4 (Par-4) gene was up-regulated after NSAID treatment. Par-4 was first isolated from prostate carcinoma cells undergoing apoptosis, and expression of Par-4 sensitized \*\*\*cancer\*\*\* cells to apoptotic stimuli. Par-4 levels were increased in cells treated with COX inhibitors such as NS-398, nimesulide, SC-58125, and sulindac sulfide. Treatment of HCA-7 cells with these agents also induced apoptotic cell death. CONCLUSIONS: The results suggest that regulation of Par-4 contributes to the proapoptotic effects of high-dose COX inhibitors (NSAIDs) by serving as a downstream mediator leading to initiation of programmed cell death.

AB . . . cells (HCA-7) and identified several genes that are regulated after treatment with NS-398, a selective COX-2 inhibitor. METHODS: Differential display \*\*\*polymerase\*\*\* chain reaction cloning techniques were used to identify genes regulated by treatment with NSAIDs and selective COX-2 inhibitors. RESULTS: A. . . was up-regulated after NSAID treatment. Par-4 was first isolated from prostate carcinoma cells undergoing apoptosis, and expression of Par-4 sensitized \*\*\*cancer\*\*\* cells to apoptotic stimuli. Par-4 levels were increased in cells treated with COX inhibitors such as NS-398, nimesulide, SC-58125, and. . .

CT . . . pharmacology  
\*Apoptosis: DE, drug effects  
Apoptosis: GE, genetics  
Blotting, Northern  
Blotting, Western  
Carrier Proteins: AN, analysis  
\*Carrier Proteins: GE, genetics  
\*\*\* Colonic Neoplasms\*\*\*  
Cyclooxygenase Inhibitors: PD, pharmacology  
DNA Fragmentation  
Gene Expression: DE, drug effects  
Gene Expression: PH, physiology  
Humans  
\*\*\* Intestinal Mucosa: CH, chemistry\*\*\*  
\*\*\*\*Intestinal Mucosa: CY, cytology\*\*\*  
\*\*\* Intestinal Mucosa: EN, enzymology\*\*\*  
\*Intracellular Signaling Peptides and Proteins  
\*Nitrobenzenes: PD, pharmacology  
Protein Kinase C: ME, metabolism  
Pyrazoles: PD, pharmacology  
. . . Support, U.S. Gov't, Non-P.H.S.  
Research Support, U.S. Gov't, P.H.S.  
\*Sulfonamides: PD, pharmacology  
Sulindac: AA, analogs & derivatives  
Sulindac: PD, pharmacology  
\*\*\* Tumor Cells, Cultured\*\*\*

RN 123653-11-2 (N-(2-cyclohexyloxy-4-nitrophenyl)methanesulfonamide);  
162054-19-5 (1-((4-methylsulfonyl)phenyl)-3-trifluoromethyl-5-(4-fluorophenyl)pyrazole); 32004-67-4 (sulindac sulfide); \*\*\*38194-50-2\*\*\*  
\*\*\* (Sulindac)\*\*\* ; 51803-78-2 (nimesulide)

=> d his

(FILE 'HOME' ENTERED AT 09:22:46 ON 14 DEC 2005)

FILE 'REGISTRY' ENTERED AT 09:22:55 ON 14 DEC 2005

E "SULINDAC"/CN 25

L1 1 S E3

FILE 'CAPLUS' ENTERED AT 09:23:34 ON 14 DEC 2005

L2 1426 S L1

L3 254068 S GASTROINTESTINAL OR ESOPHAG? OR GASTIC? OR INTESTI  
 L4 1099978 S CANCER? OR TUMOR? OR NEOPLAS? OR POLYP?  
 L5 65506 S L4 AND L3  
 L6 234 S L5 AND L2  
 L7 243958 S ORAL?  
 L8 30 S L7 AND L6  
 L9 186 S L2 (L) L4  
 L10 121 S L9 AND L3  
 L11 14 S L10 AND L7  
 L12 9 S L11 NOT PY>2002  
 L13 1945587 S LIPOM? OR MICROSPHER? OR ENCAPSULAT? OR POLYMER?  
 L14 0 S L13 AND L12  
 L15 443 S L4 AND L2  
 L16 12 S L9 AND L13  
 L17 3 S L16 NOT PY>2002

FILE 'MEDLINE' ENTERED AT 09:35:28 ON 14 DEC 2005

L18 919 S SULINDAC/CN  
 L19 1879233 S CANCER? OR TUMOR? OR NEOPLAS? OR POLYP?  
 L20 428581 S GASTROINTESTINAL OR ESOPHAG? OR GASTIC? OR INTESTI  
 L21 125328 S L19 AND L20  
 L22 175 S L21 AND L18  
 L23 407843 S LIPOSOM? OR MICROSPHER? OR ENCAPSULAT? OR POLYMER?  
 L24 8 S L23 AND L22  
 L25 6 S L24 NOT PY>2002

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	ENTRY	SINCE FILE SESSION	TOTAL
CA SUBSCRIBER PRICE		0.00	-3.65

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FILE COVERS 1907 - 14 Dec 2005 VOL 143 ISS 25  
 FILE LAST UPDATED: 13 Dec 2005 (20051213/ED)

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=> s liposom? or microspher? or encapsulat? or polymer?

48683 LIPOSOM?  
 27180 MICROSPHER?  
 55572 ENCAPSULAT?  
 1820552 POLYMER?  
 84067 POLYMD  
 84067 POLYMD  
 (POLYMD)  
 31147 POLYMG  
 326031 POLYMN  
 8505 POLYMNS  
 327118 POLYMN  
 (POLYMN OR POLYMNS)  
 1885881 POLYMER?  
 (POLYMER? OR POLYMD OR POLYMG OR POLYMN)

L26 1984458 LIPOSOM? OR MICROSPHER? OR ENCAPSULAT? OR POLYMER?

=> s l9 and l26

L27 15 L9 AND L26

=> s liposom? or microspher? or encapsulat?

48683 LIPOSOM?

27180 MICROSPHER?

55572 ENCAPSULAT?

L28 122087 LIPOSOM? OR MICROSPHER? OR ENCAPSULAT?

=> s l28 and l9

L29 11 L28 AND L9

=> s l29 not py>2002

3346380 PY>2002

L30 0 L29 NOT PY>2002

=> s l29 not py>2003

2266400 PY>2003

L31 0 L29 NOT PY>2003

=> d l29 ibib 1-4

L29 ANSWER 1 OF 11 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2005:591975 CAPLUS

DOCUMENT NUMBER: 143:53482

TITLE: Method for inhibiting the growth of gastrointestinal tract tumors

INVENTOR(S): Egilmez, Nejat K.

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 21 pp.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005147689	A1	20050707	US 2003-748003	20031230
CA 2491338	AA	20050630	CA 2004-2491338	20041223
PRIORITY APPLN. INFO.:			US 2003-748003	A 20031230

L29 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2005:14227 CAPLUS

DOCUMENT NUMBER: 142:107439

TITLE: Cardiolipin synthesis inhibitor for treatment of cardiovascular disorders, and obesity

INVENTOR(S): Jamil, Haris; Ahmad, Moghis U.; Ahmad, Imran

PATENT ASSIGNEE(S): Neopharm, Inc., USA

SOURCE: PCT Int. Appl., 48 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005000318	A2	20050106	WO 2004-US20104	20040623
WO 2005000318	A3	20050414		
WO 2005000318	B1	20050526		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW  
RV: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

L29 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2004:877933 CAPLUS

DOCUMENT NUMBER: 141:365149

TITLE: Anti-PSGL-1 antibodies and scFv fragments for  
diagnosis, prognosis and therapy of cancer,  
metastasis, autoimmune disease and inflammationINVENTOR(S): Levanon, Avigdor; Ben-Levy, Rachel; Plaksin, Daniel;  
Szanton, Esther; Hagai, Yocheved; Mar-Chaim, Hagit  
Hoch

PATENT ASSIGNEE(S): Israel

SOURCE: U.S. Pat. Appl. Publ., 49 pp.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004208877	A1	20041021	US 2003-611588	20030630
PRIORITY APPLN. INFO.:			US 2002-393491P	P 20020701

L29 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2004:856929 CAPLUS

DOCUMENT NUMBER: 141:348831

TITLE: Antibodies specific to epitopes involving cell  
rolling, metastasis and inflammation for treatment of  
tumor, restenosis, thrombosis, autoimmune disease and  
inflammationINVENTOR(S): Lazarovits, Janette; Nimrod, Abraham; Hoch, Mar-Chaim  
Hagit; Levanon, Avigdor

PATENT ASSIGNEE(S): Israel

SOURCE: U.S. Pat. Appl. Publ., 22 pp.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004202665	A1	20041014	US 2003-610843	20030630
PRIORITY APPLN. INFO.:			US 2002-393453P	P 20020701

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FULL ESTIMATED COST		23.21	96.74

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
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MOST RECENT UPDATE WEEK: 200549 &lt;200549/EW&gt;

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=&gt; s SULINDAC

L32 2826 SULINDAC

=&gt; s I32/ab

L33 9 (SULINDAC/AB)

=> s cancer? or tumor? or neoplas? or polyp?

73935 CANCER?

61948 TUMOR?

21353 NEOPLAS?

153344 POLYP?

L34 196562 CANCER? OR TUMOR? OR NEOPLAS? OR POLYP?

=> s l34 and l33

L35 7 L34 AND L33

=> s gastrointestinal or esophag? or gastic? or intestin? or colorect?

4 GASTROINTESTINAL

11126 ESOPHAG?

83 GASTIC?

38774 INTESTIN?

8423 COLORECT?

L36 47131 GASTROINTESTINAL OR ESOPHAG? OR GASTIC? OR INTESTIN?  
T?

=> s gastrointestinal or esophag? or gastic? or intestin? or colorect?

28847 GASTROINTESTINAL

9 GASTROINTESTINALS

28851 GASTROINTESTINAL

(GASTROINTESTINAL OR GASTROINTESTINALS)

11126 ESOPHAG?

83 GASTIC?

38774 INTESTIN?

8423 COLORECT?

L37 59284 GASTROINTESTINAL OR ESOPHAG? OR GASTIC? OR INTESTIN? O  
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=> s l37 and l35

L38 7 L37 AND L35

=> s liposom? or microspher? or encapsulat?

40590 LIPOSOM?

15203 MICROSPHER?

61501 ENCAPSULAT?

L39 90511 LIPOSOM? OR MICROSPHER? OR ENCAPSULAT?

=> s l39 and l38

L40 2 L39 AND L38

=> d ibib 1-2

L40 ANSWER 1 OF 2 PCTFULL COPYRIGHT 2005 Univentio on STN

ACCESSION NUMBER: 2001035956 PCTFULL ED 20020820

TITLE (ENGLISH): USE OF NSAIDs FOR THE TREATMENT OF PANCREATIC  
\*\*\*CANCER\*\*\*

TITLE (FRENCH): UTILISATION DES AINS DANS LE TRAITEMENT DU  
\*\*\*CANCER\*\*\* DU PANCREAS

INVENTOR(S): MARSHALL, Mark, Steven;

SWEENEY, Christopher, J.;

YIP-SCHNEIDER, Michelle, T.;

CROWELL, Pamela, L.

PATENT ASSIGNEE(S): ADVANCED RESEARCH AND TECHNOLOGY INSTITUTE

MARSHALL, Mark, Steven;

SWEENEY, Christopher, J.;

YIP-SCHNEIDER, Michelle, T.;

CROWELL, Pamela, L.

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER KIND DATE

WO 2001035956 A1 20010525

DESIGNATED STATES

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU  
CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN  
IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK  
MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM  
TR TT TZ UA UG US UZ VN YU ZA ZW GH GM KE LS MW MZ SD  
SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY

DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF  
CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2000-US31410 A 20001115  
PRIORITY INFO.: US 1999-60/165,543 19991115

L40 ANSWER 2 OF 2 PCTFULL COPYRIGHT 2005 Univentio on STN  
ACCESSION NUMBER: 1999049859 PCTFULL ED 20020515  
TITLE (ENGLISH): DFMO AND SULINDAC COMBINATION IN \*\*\*CANCER\*\*\*  
CHEMOPREVENTION  
TITLE (FRENCH): COMBINAISON DE DFMO ET DE SULINDAC DANS LA  
CHIMIOPREVENTION DU \*\*\*CANCER\*\*\*  
INVENTOR(S): GERNER, Eugene, W.;  
MEYSKENS, Frank, L., Jr.  
PATENT ASSIGNEE(S): THE ARIZONA BOARD OF REGENTS on behalf of THE  
UNIVERSITY OF ARIZONA;  
THE REGENTS OF THE UNIVERSITY OF CALIFORNIA;  
GERNER, Eugene, W.;  
MEYSKENS, Frank, L., Jr.  
LANGUAGE OF PUBL.: English  
DOCUMENT TYPE: Patent  
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9949859	A1	19991007

DESIGNATED STATES  
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK  
EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP  
KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL  
PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN  
YU ZA ZW GH GM KE LS MW SD SL SZ UG ZW AM AZ BY KG KZ  
MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU  
MC NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD  
TG

APPLICATION INFO.: WO 1999-US6693 A 19990326  
PRIORITY INFO.: US 1998-60/079,850 19980328

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L40 ANSWER 1 OF 2 PCTFULL COPYRIGHT 2005 Univentio on STN  
TIEN USE OF NSAIDs FOR THE TREATMENT OF PANCREATIC \*\*\*CANCER\*\*\*  
TIFR UTILISATION DES AINS DANS LE TRAITEMENT DU \*\*\*CANCER\*\*\* DU PAI  
ABEN The invention provides a method comprising the use of non-steroidal  
antiinflammatory drugs (NSAIDs), particularly \*\*\*sulindac\*\*\* or its  
analogues to treat pancreatic \*\*\*cancer\*\*\*.

DETD USE OF NSAIDs FOR THE TREATMENT OF PANCREATIC \*\*\*CANCER\*\*\*

Background of the Invention

\*\*\*Cancer\*\*\* of the pancreas ranks 'ust behind lung \*\*\*cancer\*\*\*

, colon \*\*\*cancer\*\*\*, and

breast \*\*\*cancer\*\*\* as the most common cause of death by

\*\*\*cancer\*\*\* (1). It is more

common among men, and men between the ages of 60 and 70 are most at  
risk.

The cause of pancreatic \*\*\*cancer\*\*\* is unknown.

which are not fully understood, usually is

10 significant. The average loss is about 25 pounds. Jaundice occurs if  
the \*\*\*cancer\*\*\*

blocks the common bile duct. The survival rate with pancreatic  
\*\*\*cancer\*\*\* is poor.

By the time the malignant \*\*\*tumor\*\*\* is identified, it often has  
spread (metastasized)

to other parts of the body. The median survival is little more than six.

5 Often the \*\*\*tumor\*\*\* cannot be removed by surgery, either because  
it has

invaded vital structures that cannot be removed or because it has spread  
to

distant sites. Chemotherapy and radiation therapy can be used on the

\*\*\*tumor\*\*\*,

although these treatments often are not beneficial.

Easton, PA (18th ed., 1990) at pages 1115

There is a large amount of literature on the effect of NSAIDs on

\*\*\*cancer\*\*\*,

particularly colon \*\*\*cancer\*\*\*. For example, see H. A. Weiss et al., Scand J.

in vitro, but that

indomethacin, ketoralac and NS-398, did not. Sulindac has been investigated in

combination therapy for the treatment of colon \*\*\*cancer\*\*\*. See, H. M. Verheul et al.,

Brit- J. Cancer, 79, 114 (1999); F. A. Sinicropo et al., Clin.

\*\*\*Cancer\*\*\* Res-, 2, 37

(1996); and M. Mooghen et al., J. Pathol., 116, 394 (1988).

C. P. Duffy et al., Eur. J. \*\*\*Cancer\*\*\*, 34, 1250 (1998), reported that the

cytotoxicity of certain chemotherapeutic drugs was enhanced when they were

combined with certain non-steroidal anti-inflammatory agents. The effects

observed against human lung \*\*\*cancer\*\*\* cells and human leukemia cells were highly

specific and not predictable; i.e., some combinations of NSAID and agent were

effective and some.

a PCT application (WO98/18490) on October 24,

1997, directed to a combination of a substrate for MRP, which can be an anti-

\*\*\*cancer\*\*\* drug, and a NSAID that increases the potency of the anti- \*\*\*cancer\*\*\* drug.

Therefore, a continuing need exists for methods to control

\*\*\*cancers\*\*\*, and to

increase the potency of anti- \*\*\*cancer\*\*\* drugs with relatively non-toxic agents.

Summary of the Invention

In one aspect, the present invention provides a therapeutic method to treat

pancreatic \*\*\*cancer\*\*\*, comprising administering to a mammal afflicted with

pancreatic \*\*\*cancer\*\*\* an amount of a NSAID, preferably sulindac ((Z) fluoro

methyl-1-[4-(methylsulfinyl)phenyl] methylene]-1H-Indene acetic acid), or

an analog thereof, preferably one that is a COX-2 inhibitor, effective to inhibit

the viability of pancreatic \*\*\*cancer\*\*\* cells of said mammal. The present invention

also provides a method of increasing the susceptibility of human pancreatic

\*\*\*cancer\*\*\* cells to a chemotherapeutic agent comprising contacting the cells with an

effective sensitizing amount of a NSAID, preferably sulindac, or said analog

thereof. Thus, the invention provides a therapeutic method for the treatment of a

human or other mammal afflicted with pancreatic \*\*\*cancer\*\*\*, wherein an effective

amount of an NSAID, preferably sulindac or said analog thereof is administered

to a subject afflicted with pancreatic \*\*\*cancer\*\*\* and undergoing treatment with a

5 chemotherapeutic (antineoplastic) agent.

Preferably, sulindac is administered in conjunction with one or more chemotherapeutic agents effective against pancreatic \*\*\*cancer\*\*\*

such as gemcitabine

or 5-FU.



A method of evaluating the ability of sulindac to sensitize pancreatic \*\*\*cancer\*\*\* cells to a chemotherapeutic agent is also provided. The assay method comprises: (a) isolating a first portion of pancreatic \*\*\*cancer\*\*\* cells from a human \*\*\*cancer\*\*\* patient; (b) measuring their viability; (c) administering sulindac, or said analog thereof, to said patient; (d) isolating a second portion of pancreatic \*\*\*cancer\*\*\* cells from said patient; (e) measuring the viability of the second portion of pancreatic \*\*\*cancer\*\*\* cells; and (f) comparing the viability measured in step (e) with the viability measured in step (b); wherein reduced viability in. . .

(b) and (e) are carried out in the presence of the chemotherapeutic agent, as will be the case when the pancreatic \*\*\*cancer\*\*\* cells are derived from the blood of a mammal afflicted with pancreatic \*\*\*cancer\*\*\*.

Thus, a \*\*\*cancer\*\*\* patient about to undergo, or undergoing, treatment for pancreatic \*\*\*cancer\*\*\* can be rapidly evaluated to see if he/she will benefit from concurrent chemotherapy and administration of sulindac or an analog thereof.

#### Description of the Figures

Figure 1. Photocopy of a representative immunoblot of pancreatic adenocarcinomas and matched normal tissue. Lysates were prepared from \*\*\*tumor\*\*\*

(T) specimens obtained from six patients, three with matched normal (N) tissue

(sample numbers correspond to those listed in Table 1). Lysates. . . expresses neither COX-1 or COX

Figure 2. Percent COX-2 expression in patient samples. Values of % COX-2 expression for all \*\*\*tumor\*\*\* samples, shown by solid circles, and non-nal tissue, shown by open circles, from Table I are plotted. Values for mean, median and range are indicated. The % COX-2 expression for the matched pancreatic

\*\*\*tumor\*\*\* /normal tissue sets is shown in the inset (n = 11).

Lines are drawn between the corresponding \*\*\*tumor\*\*\* values, shown by solid circles, and non-nal values, shown by the open circles. The difference in COX-2 expression between \*\*\*tumor\*\*\* and non-nal specimens was determined to be statistically significant (P = 0.004).

Figure 3. COX-2 expression in pancreatic \*\*\*tumor\*\*\* cell lines. A) COX-2 expression in human pancreatic cell lines detected by immunoblot analysis. The K-ras mutation status of each of the. . .

Figure 4. Effect of COX inhibitors on the growth of pancreatic \*\*\*tumor\*\*\* cell lines. The cell lines BxPC-3, shown by the black bars, and PaCa-2, shown by the hatched bars, were plated in the. . .

Figure 5. Prostaglandin E2 production. A) PGE2 levels in pancreatic \*\*\*tumor\*\*\* cell lines. Following incubation of exponentially growing cells with 15 gM arachidonic acid in serum-free media for one hour, PGE2 levels. . .

Figure 6 is a graph depicting the effect of a combination of sulindac and gemcitabine on the growth of pancreatic \*\*\*tumor\*\*\* cell line BxPC.

Figure 7 is a graph depicting the effect of a combination of sulindac and gemcitabine on the growth of pancreatic \*\*\*tumor\*\*\* cell line PaCa. Detailed Description of the Invention

Difficulty in achieving early diagnosis as well as the aggressive nature of pancreatic \*\*\*cancer\*\*\* contribute to the low survival rate of patients with pancreatic \*\*\*cancer\*\*\*. Since few options exist for the treatment of pancreatic \*\*\*cancer\*\*\*, it is important to identify potential targets for drug therapy. In an effort to gain more insight into pancreatic tumorigenesis, pancreatic \*\*\*tumors\*\*\* have been analyzed at the molecular level to detect genetic lesions. Activating mutations within the K-ras gene have been detected in up to 90% of pancreatic carcinomas, suggesting that activation of the Ras pathway is important in the development of pancreatic \*\*\*cancer\*\*\* (2). Experimental chemotherapeutic strategies for pancreatic \*\*\*cancer\*\*\* patients currently include drugs which target the Ras signal transduction pathway.

For example, epidemiological studies have shown that prolonged use of aspirin or other nonsteroidal anti-inflammatory drugs (NSAIDs) can reduce the risk of colon \*\*\*cancer\*\*\* by 40-50% (3). NSAIDs also inhibit chemically induced colon carcinomas in animal model systems (4). Since NSAIDs are known to inhibit cyclooxygenase, . . . esters, and growth factors (5, 6). COX-2 expression has recently been shown to be elevated in several different types of human \*\*\*cancer\*\*\*, suggesting that the presence of COX-2 correlates with \*\*\*cancer\*\*\* development (7-11). Additional studies which directly link COX-2 to carcinogenesis include observations that human colon \*\*\*cancer\*\*\* cells expressing COX-2 acquire increased invasiveness (12) and that COX-2 expressed in \*\*\*intestinal\*\*\* epithelial cells inhibits apoptosis (13). COX-2 expression in colon \*\*\*cancer\*\*\* cells has also been found to promote angiogenesis of co-cultured endothelial cells by stimulating the production of angiogenic factors (14). Furthermore, direct genetic evidence linking COX-2 to \*\*\*colorectal\*\*\* \*\*\*tumorigenesis\*\*\* was provided by a mouse model for human familial adenomatous \*\*\*polyposis\*\*\* (FA-P), an inherited condition leading to \*\*\*colorectal\*\*\* \*\*\*cancer\*\*\*; in this system, COX-2 gene knockouts and a specific COX-2 inhibitor were found to reduce the number of \*\*\*intestinal\*\*\* \*\*\*polyps\*\*\* formed (15).

The presence of oncogenic Ras has been associated with the induction of COX-2 expression in H-ras-transformed rat \*\*\*intestinal\*\*\* and mammary epithelial cells as well as in non-small cell lung cancer cell lines (16-18). To our knowledge, the association between oncogenic Ras and COX-2 expression has not been explored in vivo. The high frequency of activating mutations within the K-ras gene in pancreatic \*\*\*tumors\*\*\* should enable us to investigate the relationship between oncogenic K-ras and COX-2 expression in vivo. In the present study,

we evaluated COX-2 protein levels in primary human pancreatic adenocarcinomas. We further examined whether COX-2 expression correlated with K-ras mutation status in pancreatic \*\*\*tumors\*\*\* as well as in pancreatic \*\*\*cancer\*\*\* cell lines. In light of our data demonstrating elevated levels of COX-2 protein in primary pancreatic \*\*\*tumors\*\*\* and cell lines, we tested the effect of the COX inhibitors sulindac, indomethacin and NS-398 on cell growth and prostaglandin E2 production in human pancreatic \*\*\*tumor\*\*\* cell lines.

Cyclooxygenase-2 (COX-2) expression is upregulated in several types of human \*\*\*cancers\*\*\* and has also been directly linked to carcinogenesis. To

15 investigate the role of COX-2 in pancreatic \*\*\*cancer\*\*\*, we evaluated COX-2 protein expression in primary human pancreatic adenocarcinomas (n = 23) and matched

normal adjacent tissue (n = 11) by immunoblot analysis. COX-2 expression was found to be significantly elevated in the pancreatic \*\*\*tumor\*\*\* specimens compared to normal pancreatic tissue. To examine whether the elevated levels of COX-2

protein observed in pancreatic \*\*\*tumors\*\*\* correlated with the presence of oncogenic

K-ras, we determined the K-ras mutation status in a subset of the \*\*\*tumors\*\*\* and

corresponding non-naïve tissues. The presence of oncogenic K-ras did not correlate with the level of COX-2 protein expressed in the pancreatic adenocarcinomas analyzed. These observations were also confirmed in a panel

of human pancreatic \*\*\*tumor\*\*\* cell lines. Furthermore, in the pancreatic \*\*\*tumor\*\*\* cell line expressing the highest level of COX-2 (BxPC-3), COX-2 expression was

demonstrated to be independent of Erk1/2 Map kinase activation. The . . . lack of

correlation between COX-2 and oncogenic K-ras expression suggests that Ras

activation may not be sufficient to inducing COX-2 expression in pancreatic

\*\*\*tumor\*\*\* cells and that the aberrant activation of signaling pathways other than Ras

may be required for up-regulating COX-2 expression. We also. . . report that the

COX inhibitors sulindac, indomethacin, and NS-398 inhibited cell growth in

both COX positive (BxPC-3) and COX negative (PaCa-2) pancreatic \*\*\*tumor\*\*\*

cell lines. However, suppression of cell growth by indomethacin and NS-398

was significantly greater in the BxPC-3 cell line compared to. . . that COX-2 may play an important role in pancreatic

tumorigenesis and therefore be a promising chemotherapeutic target for the

treatment of pancreatic \*\*\*cancer\*\*\* .

10

Other NSAIDs, including indomethacin and NS-398 also the growth of pancreatic \*\*\*tumor\*\*\* cell lines, as discussed

hereinbelow, and can also be

used in the present method, alone, or preferably in combination with sulindac.

or infusion in dosages of about 500-4000 Mg/M2 /week

for up to 7 weeks/cycle for treatment of localized or metastatic pancreatic \*\*\*cancer\*\*\*

(adenocarcinoma of the pancreas). It can also be administered in conjunction

with other anti- \*\*\*cancer\*\*\* agents, such as 5-FU. See, PDR (53rd ed., 1999) at pages

1578

The effect of sulindac or NS-398 alone and in combination with gemcitabine on the growth of pancreatic \*\*\*tumor\*\*\* cells BxPC-3 and PaCa-2 was investigated. Treatment with the drug combinations inhibited the growth of both cell lines to a greater extent. . . NF-KB DNA binding activity was inhibited by parthenolide treatment. These results suggest that anti-inflammatory drugs may enhance the effectiveness of gemcitabine against pancreatic \*\*\*tumors\*\*\* .

of a prophylactic or therapeutic dose of sulindac, an analog thereof or a combination thereof, in the acute or chronic management of \*\*\*cancer\*\*\* , i.e., pancreatic cancer, will vary with the stage of the \*\*\*cancer\*\*\* , such as the solid \*\*\*tumor\*\*\* to be treated, the chemotherapeutic agent(s) or other anti- \*\*\*cancer\*\*\* therapy used, and the route of administration. The dose, and perhaps the dose frequency, will also vary according to the age, body. . .

5 chemotherapy regimen. The sulindac, in some cases, may be combined with the same carrier or vehicle used to deliver the anti- \*\*\*cancer\*\*\* chemotherapeutic agent.

sterile powders comprising the active ingredient which are adapted for the extemporaneous preparation of sterile injectable or infusible solutions or dispersions, optionally \*\*\*encapsulated\*\*\* in \*\*\*liposomes\*\*\* . In all cases, the ultimate dosage form must be sterile, fluid and stable under the conditions of manufacture and storage. The. . . like), vegetable oils, non-toxic glyceryl esters, and suitable mixtures thereof The proper fluidity can be maintained, for example, by the formation of \*\*\*liposomes\*\*\* , by the maintenance of the required particle size in the case of dispersions or by the use of surfactants. The prevention. . .

were obtained from the Indiana University Tissue Procurement Laboratory and the Cooperative Human Tissue Network (CHTN) which is funded by the National \*\*\*Cancer\*\*\* Institute. A total of 23 primary human pancreatic \*\*\*cancer\*\*\* specimens were analyzed in this study.

within 1 hour of surgical removal and subsequently stored at -80°C. Paraffin sections were prepared from a subset of the specimens. All \*\*\*tumor\*\*\* specimens used in this study were examined by a pathologist and classified as primary pancreatic adenocarcinomas.

5. Statistical Analysis. The presence of statistically significant elevation of COX-2 protein between \*\*\*cancer\*\*\* specimens and corresponding normal adjacent tissues was determined by the nonparametric signed rank test. A two-way analysis of variance (ANOVA) was used. . .

6. Cell Lines. The human pancreatic \*\*\*tumor\*\*\* cell lines (AsPC-1, BxPC-3, Capan-1, Capan-2, HPA-F-11, Hs766T, PaCa-2 and PANC-1) were obtained from the American Type Culture Collection (ATCC, Rockville, MD). . .

Undetectable levels of COX-2 protein were observed in each of the normal specimens. In contrast, COX-2 protein expression in the

pancreatic  
5 \*\*\*tumor\*\*\* tissues ranged from undetectable (sample #2 1) to slight/moderate (samples #12, 14, 20) to high levels (samples #9, 22). COX-1 protein was observed in both pancreatic \*\*\*tumor\*\*\* and normal tissues, although the level of expression was variable and not consistently elevated in the \*\*\*tumor\*\*\* specimens (Figure 1). Similar levels of p21<sup>+</sup> and actin expression were found in both the \*\*\*tumor\*\*\* and corresponding normal tissues (Figure 1).

narrower range (0-3%) of COX-2 expression in the normal tissues. Both the mean and median COX-2 expression were higher in the \*\*\*tumor\*\*\* samples, suggesting that COX-2 expression is elevated in pancreatic adenocarcinomas compared to normal tissue. The difference in COX-2 expression between the pancreatic \*\*\*tumor\*\*\* and corresponding normal tissue was determined to be statistically significant ( $P = 0.004$ ) (Figure 2, inset).

less than 5% respectively, which corresponds closely with visual detection in the immunoblots. According to these criteria, 6 out of 11 (55%) \*\*\*tumor\*\*\* samples in the matched tissue sets were COX-2 positive. Similarly, 13 out of the 23 (56%) total \*\*\*tumor\*\*\* specimens analyzed were COX-2 positive; in contrast, all the normal tissue samples ( $n = 11$ ) were COX-2 negative.

Immunohistochemical staining of the pancreatic \*\*\*tumor\*\*\* specimens demonstrated that COX-2 expression was localized to the carcinoma cells and was not detectable in the stromal compartment of the \*\*\*tumors\*\*\* (Figure 3).

#### Example 2

COX-2 expression and K-ras mutation in pancreatic \*\*\*tumors\*\*\* and cell lines

To determine if COX-2 expression levels correlated with the K-ras mutation status of the \*\*\*tumors\*\*\*, genomic DNA was isolated from a subset of the tissue specimens and screened for the presence of K-ras mutations at codon . . .

the normal tissues analyzed were wild-type at codon 12 (GGT = Gly) and codon 13 (GGC = Gly). Of the 13 pancreatic \*\*\*cancer\*\*\* specimens analyzed, one specimen had a mutation at codon 13 whereas 10 samples were mutated at codon 12, corresponding to a K-ras. . . extent of COX-2 protein expression. For example, some samples expressed high levels of COX-2 protein and possessed a mutation in K-ras (i.e., \*\*\*tumor\*\*\* samples #9, 16 and 22); however, other samples which had mutated K-ras expressed little or no COX-2 protein (i.e., \*\*\*tumor\*\*\* samples #3, 17, 18, 19, and 21).

with known K-ras mutation status (25, 26). Both the frequency and variability in the quantity of COX-2 expressed in the pancreatic \*\*\*tumor\*\*\* cell lines reflected our findings in the primary pancreatic adenocarcinomas. Of the eight human pancreatic \*\*\*tumor\*\*\* cell lines analyzed, only three of the seven cell lines expressing oncogenic K-ras exhibited detectable levels of COX-2 protein

(Capan-1, Capan-2 and. . . (Figure 4B). Taken together, our results suggest that activation of the Ras pathway is not sufficient for mediating COX-2 upregulation in pancreatic \*\*\*tumor\*\*\* cells. We also compared the level of COX-2 expression in three hamster pancreatic cell lines, The D27/K-ras and B 12/13 transformed cell. . . parental line (Figure 4Q). These results confirm our conclusion that Ras activation alone is not sufficient for upregulating COX-2 expression in pancreatic \*\*\*cancer\*\*\* cells and suggest that additional events which occur following exposure to chemical carcinogens may be required.

To examine whether COX-2 expression could be induced in the human pancreatic \*\*\*cancer\*\*\* cell lines, four cell lines were serum-starved and subsequently treated with 10% FCS for various time periods (Figure 4D). In. . .

is activated (unpublished observations), again demonstrating that Erk 1/2 activation is not sufficient for inducing COX-2 expression in the COX negative pancreatic \*\*\*tumor\*\*\* cells. We observed similar results upon treating the cell lines with the \*\*\*tumor\*\*\* promoter, PMA (unpublished observations).

### Example 3

Treatment of pancreatic \*\*\*tumor\*\*\* cell lines with cyclooxygenase inhibitors

The COX positive human pancreatic \*\*\*tumor\*\*\* cell lines, BxPC-3, and the

COX negative cell line, PaCa-2, were treated with the COX inhibitors sulindac, indomethacin, or NS Sulindac and. . . was measured after three days of treatment (Figure 5). All three inhibitors were found to suppress cell growth in both pancreatic \*\*\*tumor\*\*\* cell lines

in a dose-dependent manner. However, indomethacin and NS-398 were found to inhibit cell growth to a greater extent in the. . .

To evaluate the functional activity of COX-2 in the human pancreatic \*\*\*tumor\*\*\* cell lines, prostaglandin E<sub>2</sub> (PGE<sub>2</sub>) production was measured by enzyme immunoassay (Figure 6A). PGE<sub>2</sub> production was elevated in the BxPC-3, Capan-1, Capan-2. . .

These data demonstrate that the combination of sulindac and gemcitabine is more effective than either compound alone in pancreatic \*\*\*tumor\*\*\* cells.

as well as inflammatory agents (5, 6, 29). Recent studies have shown that COX-2 expression is upregulated in a variety of human \*\*\*cancers\*\*\*, including colon, lung, gastric, pancreatic and

\*\*\*esophageal\*\*\* (7-11). In the present study, we report that elevated levels of COX-2 protein are expressed in human pancreatic \*\*\*tumors\*\*\* compared to barely detectable levels in the matched non-tumoral pancreatic tissue, suggesting that increased

expression of COX-2 protein correlates with pancreatic tumorigenesis. Our

results confirm a recent report demonstrating upregulation of COX-2 RNA and

protein in pancreatic \*\*\*tumors\*\*\* and localization of COX-2 in malignant epithelial

cells (11). An earlier study demonstrated that the expression of group

phospholipase A<sub>2</sub>,. . . phospholipids, was higher in pancreatic ductal adenocarcinomas

compared to normal pancreatic tissue (30). In addition, the development of N-

nitrosobis(2-oxopropyl)amine (BOP)-initiated pancreatic \*\*\*tumors\*\*\* in hamsters was inhibited by the administration of two prostaglandin synthesis inhibitors, phenylbutazone and indomethacin (31). Together with our observations in . . . that increased prostaglandin production due to the increased expression of COX-2 may be an important event in the multi-step progression towards pancreatic \*\*\*tumor\*\*\* formation.

as well as prostaglandin E2 were detected in Ras-transformed mammary epithelial cells (C57/MG) cells (17). In human non-small cell lung \*\*\*cancer\*\*\* (NSCLC) cell lines expressing oncogenic K-Ras, increased PGE2 production was mediated by constitutively high expression of cytosolic, phospholipase A, and COX-2 compared. . . the expression of detectable levels of COX-2 protein. A possible explanation for the lack of COX-2 expression in a subset of the \*\*\*tumors\*\*\* with oncogenic Ras is that Erk1/2 activity may be down-regulated in pancreatic carcinomas (26). Moreover, even in the two pancreatic \*\*\*tumor\*\*\* samples which did show elevated levels of activated Erk1/2 (samples #4 and 21, data not shown), only low levels of COX-2. . . in the present study, suggesting that Erk1/2 activation alone is not sufficient for inducing COX-2 expression. These findings suggest that within the \*\*\*tumor\*\*\* environment, the presence of oncogenic K-ras does not directly result in increased COX-2 expression in pancreatic \*\*\*cancer\*\*\*.

Similar conclusions were also reached upon analysis of pancreatic \*\*\*cancer\*\*\* cell lines, which were examined since they represent a homogenous population of cells as opposed to primary \*\*\*tumor\*\*\* tissue which is heterogenous. Despite activating K-ras mutations in seven out of the eight lines, only three of the lines with mutated. . . of COX-2 expression. Activation of other signaling pathways in addition to Ras may cooperate to determine the extent of COX-2 expression in \*\*\*cancer\*\*\* cells. Such pathways may include the p38 mitogen-activated protein kinase which has been reported to regulate the induction of COX-2 in lipopolysaccharide-treated. . . the cell type as well as the stimulus. Further experiments will be required to delineate which signaling pathways are function in pancreatic \*\*\*tumor\*\*\* cells.

expressing cell lines. These data suggest that the COX inhibitors exert their inhibitory effects by both COX/PGE<sub>2</sub>-dependent and -independent pathways in pancreatic \*\*\*tumor\*\*\* cell lines.

The detection of elevated levels of COX-2 in a variety of human \*\*\*cancers\*\*\* combined with the chemopreventative effect of NSAIDs in colon \*\*\*cancer\*\*\*

10 demonstrate that COX-2 is an important participant in carcinogenesis. The reported biological consequences of COX-2 upregulation include inhibition of apoptosis (13), increased metastatic potential (12) and promotion of angiogenesis (14). These events may contribute to cell transformation and

\*\*\*tumor\*\*\* progression.

COX-2 expression was noticeably elevated in 55% of the patient pancreatic  
\*\*\*tumor\*\*\* samples analyzed, identifying COX-2 as a new target for chemotherapy.

These results demonstratancy the ability of COX inhibitors to inhibit pancreatic  
\*\*\*tumor\*\*\* cell growth and PGE<sub>2</sub> production in vitro indicate that NSAIDs may be effective in the treatment of pancreatic \*\*\*cancer\*\*\* patients, for whom few treatment options currently exist. COX-2 expression is also useful as a prognostic or diagnostic tool.

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TABLE 1. Analysis of Patient Samples

Tissue Sample	Tissue Type	% COX-2	b %	***Cancer***	K-ras
1	pancreatic adenocarcinoma	7.0	10	WT	
2	pancreatic adenocarcinoma	2.0	95		
3	pancreatic adenocarcinoma	0.2	15	GGC to CG	
4	pancreatic adenocarcinoma	3.6		N normal	0.1 -
12	pancreatic adenocarcinoma	1	15		
14	pancreatic adenocarcinoma	31	ND		

Tissue Sample	a Tissue Type	% COX-2	b %	***Cancer***	K-ras
1	5 pancreatic adenocarcinoma	7.8	25	GGT to	
15	N normal	4.3	-	1	
1	6 pancreatic adenocarcinoma	66	35	GGT to	
16	N non-nal.				

c The percent \*\*\*cancer\*\*\* was determined by visualization following hematoxylin/eosin staining of slides prepared from paraffin sections.

CLAIM 1. A method of reducing the viability of pancreatic \*\*\*cancer\*\*\* cells comprising contacting the \*\*\*cancer\*\*\* cells with an effective amount of an NSAID.

2 A method of increasing the susceptibility of mammalian pancreatic \*\*\*cancer\*\*\* cells to a chemotherapeutic agent comprising contacting the cells with an effective sensitizing amount of an NSAID.

4 The method of claim 1 or 2 wherein the mammalian \*\*\*cancer\*\*\* cells are human \*\*\*cancer\*\*\* cells.

5 The method of claim 3 wherein the sulindac or the analog thereof is administered to a human \*\*\*cancer\*\*\* patient.

6 The method of claim 5 wherein the \*\*\*cancer\*\*\* patient is undergoing



treatment with a chemotherapeutic agent.

9 A method of evaluating the ability of sulindac or an analog thereof that is a COX-2 inhibitor to sensitize pancreatic \*\*\*cancer\*\*\* cells to a chemotherapeutic agent comprising:  
(a) isolating a first portion of pancreatic \*\*\*cancer\*\*\* cells from a human pancreatic \*\*\*cancer\*\*\* patient;  
(b) measuring their viability;  
(c) administering sulindac or the analog thereof to said patient;  
(d) isolating a second portion of pancreatic \*\*\*cancer\*\*\* cells from said patient;  
(e) measuring the viability of the second portion of pancreatic \*\*\*cancer\*\*\* cells; and  
(f) comparing the viability measured in step (e) with the viability measured in step (b); wherein reduced viability in step (e) indicates. . .

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lo-  
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\*\*\*TUMOR\*\*\* NORMAL  
(n--23)  
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nwan = 15.2 +/- 24.9% mcan 0.83 +/- 1.3%  
v2mge = 0 - 93% map 0. . . Sulindac IndometIL NS-398  
% inhibition: 0 07 90 F957 98 759 86  
/8

Effect of Sulindac + Gemcitabine on the growth of the pancreatic \*\*\*tumor\*\*\* cell line, BxPC-3 (day 3)

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75 -  
1,100+ e  
50 - T  
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sul, 500 + Gem  
0 5 10 15 20. . . and Technology Institute, Inc.

Marshall, Mark Steven  
Sweeney, Christopher J.  
Yip-Schneider, Michele T.  
Crowell, Pamela L.

10<120> Use of NSAIDs for the treatment of pancreatic \*\*\*cancer\*\*\*

<130> 740.018WO1  
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 <210> 2  
 30<211>. . . search (name of data base and, where practical, search terms used)  
 EPO-Internal, WPI Data, PAJ., CHEM ABS Data, MEDLINE, EMBASE, BIOSIS,  
 \*\*\*CANCERLIT\*\*\*  
 C. DOCUMENTS CONSIDERED TO BE RELEVANT  
 Category Citation of document, with indication, where appropriate, of the relevant passages Relevant to claim No.  
 PqX SWEENEY J. ET AL.: INHIBITION OF CELL 1-11  
 GROWTH IN PANCREATIC \*\*\*TUMOR\*\*\* CELLS BY  
 ANTI-INFLAMMATORY DRUGS11  
 PROCEEDINGS OF THE AMERICAN ASSOCIATION  
 FOR \*\*\*CANCER\*\*\* RESEARCH,  
 vol. 41, March 2000 (2000-03),, page 527  
 XPOO2164391  
 USA  
 ABSTRACT #3358  
 abstract  
 Further documents are listed in the continuation of box C. Patent family members. . . passages Relevant to claim NO.  
 PqX MARSHALL M.S. ET AL.: SUPPRESSION OF 1-11  
 PANCREATIC DUCTAL ADENOCARCINOMA GROWTH BY  
 SULINDACH  
 PROCEEDINGS OF THE AMERICAN ASSOCIATION  
 FOR \*\*\*CANCER\*\*\* RESEARCH,  
 vol. 41, March 2000 (2000-03), page 526  
 XPOO2164392  
 USA  
 ABSTRACT #3349  
 abstract  
 P9X T.YIP-SCHNEIDER M. ET AL.: COX-2 1-11  
 EXPRESSION IN HUMAN PANCREATIC  
 ADENOCARCINOMAS11  
 CARCINOGENESIS,  
 vol. 21, no. 2,, . . . XPOO0984815  
 the whole document  
 X MOLINA M, ET AL.: INCREASED COX-2 1-11  
 EXPRESSION IN HUMAN PANCREATIC CARCINOMAS  
 AND CELL LINES: GROWTH INHIBITION BY  
 NONSTEROIDAL ANTI-INFLAMMATORY DRUGS11  
 \*\*\*CANCER\*\*\* RESEARCH,  
 vol. 59, no. 17, September 1999 (1999-09),  
 pages 4356-4362, XPOO0984712  
 the whole document  
 X WO 99 49859 A (THE ARIZONA BOARD OF 1-698  
 REGENTS). . .

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=> s microspher?

L1 15203 MICROSPHER?

=> s l1/ti

L2 343 (MICROSPHER?/TI)

=> s l1/ab

L3 990 (MICROSPHER?/AB)

=> s l2 or l3

L4 1026 L2 OR L3

=> s polyanhydride

1149 POLYANHYDRIDE

5384 POLYANHYDRIDES

L5 6164 POLYANHYDRIDE

(POLYANHYDRIDE OR POLYANHYDRIDES)

=> s sulindac

L6 2826 SULINDAC

=> s l6 and l4

L7 16 L6 AND L4

=> s l7 and l5

L8 3 L7 AND L5

=> d ibib 1-3

L8 ANSWER 1 OF 3 PCTFULL COPYRIGHT 2005 Univentio on STN  
ACCESSION NUMBER: 2005081825 PCTFULL ED 20050914 EW 200536  
TITLE (ENGLISH): ABUSE RESISTANT OPIOID TRANSDERMAL DELIVERY DE'  
CONTAINING OPIOID ANTAGONIST \*\*\*MICROSPHERES\*\*\*  
TITLE (FRENCH): DISPOSITIF DE DISTRIBUTION TRANSDERMIQUE D'OPIOID  
EMPECHANT UNE UTILISATION ABUSIVE ET CONTENANT DES  
\*\*\*MICROSPHERES\*\*\* D'ANTAGONISTES D'OPIOIDES  
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10018\$, US

LANGUAGE OF FILING: English

LANGUAGE OF PUBL.: English

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 2005081825	A2	20050909
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DESIGNATED STATES

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RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2005-US4741 A 20050215

PRIORITY INFO.: US 2004-60/547,196 20040223

L8 ANSWER 2 OF 3 PCTFULL COPYRIGHT 2005 Univentio on STN

ACCESSION NUMBER: 2004052339 PCTFULL ED 20040630 EW 200426

TITLE (ENGLISH): PH TRIGGERED TARGETED CONTROLLED RELEASE SYS

TITLE (FRENCH): SYSTEMES DE LIBERATION CONTROLEE CIBLEE A DECLI  
FONCTION DU PH

INVENTOR(S): SHEFER, Adi, 14 Jason Drive, East Brunswick, NJ 08816,  
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LANGUAGE OF FILING: English

LANGUAGE OF PUBL.: English

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 2004052339	A1	20040624
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DESIGNATED STATES

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RW (EPO): AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LU  
MC NL PT RO SE SI SK TR

RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2003-US26142 A 20030821

PRIORITY INFO.: US 2002-10/315,801 20021209

L8 ANSWER 3 OF 3 PCTFULL COPYRIGHT 2005 Univentio on STN

ACCESSION NUMBER: 1996040090 PCTFULL ED 20020514

TITLE (ENGLISH): METHOD FOR REDUCING OR PREVENTING POST-SURGIC  
ADHESION FORMATION USING 5-LIPOXYGENASE INHIBITORS

TITLE (FRENCH): PROCEDE POUR LA REDUCTION OU LA PREVENTION DE  
FORMATION D'ADHERENCES POST-CHIRURGICALES A L'AIDE  
D'INHIBITEURS DE 5-LIPOXYDASE

INVENTOR(S): RODGERS, Kathleen, Elizabeth;  
diZEREKA, Gere, Stodder

PATENT ASSIGNEE(S): UNIVERSITY OF SOUTHERN CALIFORNIA

LANGUAGE OF PUBL.: English

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 9640090	A1	19961219
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DESIGNATED STATES

W: AU CA JP AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL  
PT SE

APPLICATION INFO.: WO 1996-US8216 A 19960531

PRIORITY INFO.: US 1995-8/473,183 19950607

=> d his

(FILE 'HOME' ENTERED AT 08:21:20 ON 15 DEC 2005)

FILE 'PCTFULL' ENTERED AT 08:21:30 ON 15 DEC 2005

L1	15203 S MICROSPHER?
L2	343 S L1/TI
L3	990 S L1/AB
L4	1026 S L2 OR L3
L5	6164 S POLYANHYDRIDE
L6	2826 S SULINDAC
L7	16 S L6 AND L4
L8	3 S L7 AND L5

=> s l7 and polymer

	158886 POLYMER
	122941 POLYMERS
	189307 POLYMER
	(POLYMER OR POLYMERS)
L9	16 L7 AND POLYMER

=> s l7 not py>2002

	339965 PY>2002
L10	12 L7 NOT PY>2002

=> s cancer? or tumor? or cancer?

	73935 CANCER?
	61948 TUMOR?
	73935 CANCER?
L11	90719 CANCER? OR TUMOR? OR CANCER?

=> s l11 and l12

L12 NOT FOUND

The L-number entered could not be found. To see the definition of L-numbers, enter DISPLAY HISTORY at an arrow prompt (=>).

=> s l11 and l10

L12	12 L11 AND L10
-----	----------------

=> s polymeric

	89217 POLYMERIC
	178 POLYMERICS
L13	89307 POLYMERIC
	(POLYMERIC OR POLYMERICS)

=> s l13 or l9

L14	89310 L13 OR L9
-----	-----------------

=> s polymer

	158886 POLYMER
	122941 POLYMERS
L15	189307 POLYMER
	(POLYMER OR POLYMERS)

=> s l15 or l13

L16	208045 L15 OR L13
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=> s l16 and l7

L17	16 L16 AND L7
-----	---------------

=> s l17 not py>2001

443951 PY>2001  
L18 11 L17 NOT PY>2001

=> d ibib 1-5

L18 ANSWER 1 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN  
ACCESSION NUMBER: 2001072281 PCTFULL ED 20020822  
TITLE (ENGLISH): \*\*\*MICROSPHERES\*\*\* FOR ACTIVE EMBOLIZATION  
TITLE (FRENCH): \*\*\*MICROSPHERES\*\*\* PERMETTANT UNE EMBOLISATIC  
ACTIVE  
INVENTOR(S): VOGEL, Jean-Marie;  
BOSCHETTI, Egisto  
PATENT ASSIGNEE(S): BIOSPHERE MEDICAL INC.;  
VOGEL, Jean-Marie;  
BOSCHETTI, Egisto  
DOCUMENT TYPE: Patent  
PATENT INFORMATION:  
NUMBER KIND DATE  
-----  
WO 2001072281 A2 20011004  
DESIGNATED STATES  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR  
CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL  
IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG  
MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ  
TM TR TT TZ UA UG US UZ VN YU ZA ZW GH GM KE LS MW MZ  
SD SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH  
CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ  
CF CG CI CM GA GN GW ML MR NE SN TD TG  
APPLICATION INFO.: WO 2001-US9619 A 20010323  
PRIORITY INFO.: US 2000-60/191,899 20000324

L18 ANSWER 2 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN  
ACCESSION NUMBER: 2001072280 PCTFULL ED 20020822  
TITLE (ENGLISH): \*\*\*MICROSPHERES\*\*\* FOR GENE THERAPY  
TITLE (FRENCH): COMPOSITIONS ET METHODES POUR THERAPIE GENIQU  
INVENTOR(S): VOGEL, Jean-Marie;  
BOSCHETTI, Egisto  
PATENT ASSIGNEE(S): BIOSPHERE MEDICAL INC.;  
VOGEL, Jean-Marie;  
BOSCHETTI, Egisto  
DOCUMENT TYPE: Patent  
PATENT INFORMATION:  
NUMBER KIND DATE  
-----  
WO 2001072280 A2 20011004  
DESIGNATED STATES  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR  
CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL  
IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG  
MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ  
TM TR TT TZ UA UG US UZ VN YU ZA ZW GH GM KE LS MW MZ  
SD SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH  
CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ  
CF CG CI CM GA GN GW ML MR NE SN TD TG  
APPLICATION INFO.: WO 2001-US9618 A 20010323  
PRIORITY INFO.: US 2000-60/191,902 20000324

L18 ANSWER 3 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN  
ACCESSION NUMBER: 2001070291 PCTFULL ED 20020822  
TITLE (ENGLISH): INJECTABLE \*\*\*MICROSPHERES\*\*\* FOR DERMAL  
AUGMENTATION AND TISSUE BULKING  
TITLE (FRENCH): \*\*\*MICROSPHERES\*\*\* INJECTABLES DESTINEES A  
L'AUGMENTATION DERMIQUE ET AU GONFLEMENT TISSULAIRE  
INVENTOR(S): VOGEL, Jean-Marie;  
THOMAS, Richard;  
BOSCHETTI, Egisto  
PATENT ASSIGNEE(S): BIOSPHERE MEDICAL, INC.  
DOCUMENT TYPE: Patent  
PATENT INFORMATION:  
NUMBER KIND DATE  
-----  
WO 2001070291 A2 20010927  
DESIGNATED STATES

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR  
CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL  
IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG  
MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ  
TM TR TT TZ UA UG UZ VN YU ZA ZW GH GM KE LS MW MZ SD  
SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY  
DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF  
CG CI CM GA GN GW ML MR NE SN TD TG  
APPLICATION INFO.: WO 2001-US8529 A 20010315  
PRIORITY INFO.: US 2000-09/528,991 20000320

L18 ANSWER 4 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN  
ACCESSION NUMBER: 2001070289 PCTFULL ED 20020822  
TITLE (ENGLISH): INJECTABLE AND SWELLABLE \*\*\*MICROSPHERES\*\*\* FC  
TISSUE BULKING  
TITLE (FRENCH): \*\*\*MICROSPHERES\*\*\* INJECTABLES, SUSCEPTIBLES DI  
FOISSONNEMENT, VISANT A FAIRE GONFLER UN TISSU  
INVENTOR(S): VOGEL, Jean-Marie;  
BOSCHETTI, Egisto  
PATENT ASSIGNEE(S): BIOSPHERE MEDICAL, INC.  
DOCUMENT TYPE: Patent  
PATENT INFORMATION:  
NUMBER KIND DATE  
-----  
WO 2001070289 A2 20010927

DESIGNATED STATES  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR  
CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL  
IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG  
MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ  
TM TR TT TZ UA UG UZ VN YU ZA ZW GH GM KE LS MW MZ SD  
SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY  
DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF  
CG CI CM GA GN GW ML MR NE SN TD TG  
APPLICATION INFO.: WO 2001-US8405 A 20010315  
PRIORITY INFO.: US 2000-09/528,989 20000320

L18 ANSWER 5 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN  
ACCESSION NUMBER: 2000024378 PCTFULL ED 20020515  
TITLE (ENGLISH): COMPOSITIONS OF \*\*\*MICROSPHERES\*\*\* FOR WOUND  
HEALING  
TITLE (FRENCH): COMPOSITIONS A BASE DE \*\*\*MICROSPHERES\*\*\* DES  
AU TRAITEMENT DES BLESSURES  
INVENTOR(S): RITTER, Vladimir;  
RITTER, Marina  
PATENT ASSIGNEE(S): POLYHEAL LTD;  
RITTER, Vladimir;  
RITTER, Marina  
LANGUAGE OF PUBL.: English  
DOCUMENT TYPE: Patent  
PATENT INFORMATION:  
NUMBER KIND DATE  
-----  
WO 2000024378 A1 20000504

DESIGNATED STATES  
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE  
ES FI GB GE GH GM HU ID IL IS JP KE KG KP KR KZ LC LK  
LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD  
SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW GH GM  
KE LS MW SD SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE  
CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ  
CF CG CI CM GA GN GW ML MR NE SN TD TG  
APPLICATION INFO.: WO 1998-IB1838 A 19981023

=> d ibib 6-10

L18 ANSWER 6 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN  
ACCESSION NUMBER: 1998051284 PCTFULL ED 20020514  
TITLE (ENGLISH): NOVEL ACOUSTICALLY ACTIVE DRUG DELIVERY SYSTEM  
TITLE (FRENCH): NOUVEAUX SYSTEMES D'ADMINISTRATION DE MEDICAMI  
ACTIVES PAR UN PROCEDE ACOUSTIQUE  
INVENTOR(S): UNGER, Evan, C.  
PATENT ASSIGNEE(S): IMARX PHARMACEUTICAL CORP.



LANGUAGE OF PUBL.: English

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 9851284	A1	19981119
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DESIGNATED STATES

W: AU BR CA CN JP KR NZ AT BE CH CY DE DK ES FI FR GB GR  
IE IT LU MC NL PT SE

APPLICATION INFO.: WO 1998-US9569 A 19980512

PRIORITY INFO.: US 1997-60/046,379 19970513

US 1998-9/075,343 19980511

L18 ANSWER 7 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN

ACCESSION NUMBER: 1996040090 PCTFULL ED 20020514

TITLE (ENGLISH): METHOD FOR REDUCING OR PREVENTING POST-SURGIC  
ADHESION FORMATION USING 5-LIPOXYGENASE INHIBITORS

TITLE (FRENCH): PROCEDE POUR LA REDUCTION OU LA PREVENTION DE  
FORMATION D'ADHERENCES POST-CHIRURGICALES A L'AIDE  
D'INHIBITEURS DE 5-LIPOXYDASE

INVENTOR(S): RODGERS, Kathleen, Elizabeth;  
diZEREGA, Gere, Stodder

PATENT ASSIGNEE(S): UNIVERSITY OF SOUTHERN CALIFORNIA

LANGUAGE OF PUBL.: English

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 9640090	A1	19961219
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DESIGNATED STATES

W: AU CA JP AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL  
PT SE

APPLICATION INFO.: WO 1996-US8216 A 19960531

PRIORITY INFO.: US 1995-8/473,183 19950607

L18 ANSWER 8 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN

ACCESSION NUMBER: 1995015118 PCTFULL ED 20020514

TITLE (ENGLISH): GAS \*\*\*MICROSPHERES\*\*\* FOR TOPICAL AND SUBCUT/  
APPLICATION

TITLE (FRENCH): \*\*\*MICROSPHERES\*\*\* GAZEUSES POUR APPLICATION  
TOPIQUE ET SOUS-CUTANEE

INVENTOR(S): UNGER, Evan, C.;  
MATSUNAGA, Terry;  
YELLOWHAIR, David

PATENT ASSIGNEE(S): UNGER, Evan, C.;  
MATSUNAGA, Terry;  
YELLOWHAIR, David

LANGUAGE OF PUBL.: English

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 9515118	A1	19950608
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DESIGNATED STATES

W: AU CA CN JP AT BE CH DE DK ES FR GB GR IE IT LU MC NL  
PT SE

APPLICATION INFO.: WO 1994-US13817 A 19941130

PRIORITY INFO.: US 1993-8/159,674 19931130

US 1993-8/159,687 19931130

US 1993-8/160,232 19931130

US 1994-8/307,305 19940916

US 1994-8/346,426 19941129

L18 ANSWER 9 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN

ACCESSION NUMBER: 1994028874 PCTFULL ED 20020513

TITLE (ENGLISH): NOVEL THERAPEUTIC DELIVERY SYSTEMS

TITLE (FRENCH): NOUVEAU SYSTEME D'ADMINISTRATION DE PRODUITS  
THERAPEUTIQUES

INVENTOR(S): UNGER, Evan, C.;  
FRITZ, Thomas, A.;  
MATSUNAGA, Terry;  
RAMASWAMI, VaradaRajan;  
YELLOWHAIR, David;  
WU, Guanli

PATENT ASSIGNEE(S): UNGER, Evan, C.;  
FRITZ, Thomas, A.;  
MATSUNAGA, Terry;  
RAMASWAMI, VaradaRajan;  
YELLOWHAIR, David;  
WU, Guanli

LANGUAGE OF PUBL.: English

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 9428874	A1	19941222
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DESIGNATED STATES

W: AU CA CN JP AT BE CH DE DK ES FR GB GR IE IT LU MC NL  
PT SE

APPLICATION INFO.: WO 1994-US5633 A 19940519

PRIORITY INFO.: US 1993-8/076,250 19930611

US 1993-8/159,674 19931130

US 1993-8/159,687 19931130

US 1993-8/160,232 19931130

L18 ANSWER 10 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN

ACCESSION NUMBER: 1994028873 PCTFULL ED 20020513

TITLE (ENGLISH): NOVEL THERAPEUTIC DRUG DELIVERY SYSTEMS

TITLE (FRENCH): NOUVEAUX SYSTEMES D'ADMINISTRATION DE MEDICAMI

INVENTOR(S): UNGER, Evan, C.;

FRITZ, Thomas, A.;

MATSUNAGA, Terry;

RAMASWAMI, VaradaRajan;

YELLOWHAIR, David;

WU, Guanli

PATENT ASSIGNEE(S): UNGER, Evan, C.;

FRITZ, Thomas, A.;

MATSUNAGA, Terry;

RAMASWAMI, VaradaRajan;

YELLOWHAIR, David;

WU, Guanli

LANGUAGE OF PUBL.: English

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 9428873	A1	19941222
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DESIGNATED STATES

W: AU CA CN JP AT BE CH DE DK ES FR GB GR IE IT LU MC NL  
PT SE

APPLICATION INFO.: WO 1994-US5620 A 19940512

PRIORITY INFO.: US 1993-8/076,250 19930611

=> d kwic 10

L18 ANSWER 10 OF 11 PCTFULL COPYRIGHT 2005 Univentio on STN

ABEN Therapeutic drug delivery sytems comprising gas-filled

\*\*\*microspheres\*\*\* comprising a therapeutic

are described. Methods for employing such \*\*\*microspheres\*\*\* in

therapeutic drug delivery applications are

also provided. Drug delivery systems comprising gas-filled liposomes

having encapsulated therein a

drug are. . .

ABFR Systemes d'administration de medicaments au moyen de

\*\*\*microspheres\*\*\* remplies d'un gaz a effet

therapeutique, et methodes d'utilisation associees. Sont preconises des

systemes d'administration a

base de liposomes remplis. . .

DETD . . . deliver genetic material to

5 living cells. These mechanisms include techniques such as

calcium phosphate precipitation and electroporation, and

carriers such as cationic \*\*\*polymers\*\*\* and aqueous-filled

liposomes. These methods have all been relatively

ineffective in vivo and only of limited use for cell culture

transfection. None of. . .

. . .

such as ganglioside GM1  
and GM2; glucolipids; sulfatides; glycosphingolipids;

- 14 -

phosphatidic acid; palmitic acid; stearic acid; arachidonic  
acid; oleic acid; lipids bearing \*\*\*polymers\*\*\* such as  
polyethyleneglycol, chitin, hyaluronic acid or  
polyvinylpyrrolidone; lipids bearing sulfonated mono-, di-,  
5 oligo- or polysaccharides; cholesterol, cholesterol sulfate  
and cholesterol hemisuccinate; tocopherol. . .

microsphere. Preferably, this  
non-cationic lipid is dipalmitoylphosphatidylcholine,

- 15 -

dipalmitoylphosphatidylethanolamine or dioleoylphosphatidyl-  
ethanolamine. In lieu of cationic lipids as described above,  
lipids bearing cationic \*\*\*polymers\*\*\* such as polylysine or  
polyarginine may also be used to construct the microspheres  
5 and afford binding of a negatively charged therapeutic,. . .

to

carbohydrates and their phosphorylated and sulfonated  
derivatives; polyethers, preferably with molecular weight  
ranges between 400 and 8000; di- and trihydroxy alkanes and  
their \*\*\*polymers\*\*\*, preferably with molecular weight ranges  
between 800 and 8000. Emulsifying and/or solubilizing agents  
may also be used in conjunction with lipids or. . .

methicillin, nafcillin, oxacillin, penicillin G, penicillin  
V, ticarcillin rifampin and tetracycline; antiinflammatories  
such as diflunisal, ibuprofen, indomethacin, meclofenamate,  
mefenamic acid, naproxen, oxyphenbutazone, phenylbutazone,  
piroxicam, \*\*\*sulindac\*\*\*, tolmetin, aspirin and salicylates;  
20 antiprotozoans such as chloroquine, hydroxychloroquine,  
metronidazole, quinine and meglumine antimonate;  
antirheumatics such as penicillamine; narcotics such as  
paregoric; opiates. . .

DNA and analogs thereof, such as  
20 phosphorothioate and phosphorodithioate  
oligodeoxynucleotides. Additionally, the genetic material  
may be combined, for example, with proteins or other  
\*\*\*polymers\*\*\* .

form the microspheres include, for example, proteins  
such as albumin, synthetic peptides such as polyglutamic  
acid, and linear and branched oligomers and \*\*\*polymers\*\*\* of  
- 25 -

galactose, glucose and other hexosaccharides and \*\*\*polymers\*\*\*  
derived from phosphorylated and sulfonated pentose and hexose  
sugars and sugar alcohols. Carbohydrate \*\*\*polymers\*\*\* such as  
alginic acid, dextran, starch and HETA starch may also be  
5 used. Other natural \*\*\*polymers\*\*\*, such as hyaluronic acid, may  
be utilized. Synthetic \*\*\*polymers\*\*\* such as polyethyleneglycol,  
polyvinylpyrrolidone, polylactide, polyethyleneimines (linear  
and branched), polyionenes or polyiminocarboxylates may also  
be employed.

=> d his

(FILE 'HOME' ENTERED AT 08:21:20 ON 15 DEC 2005)

FILE 'PCTFULL' ENTERED AT 08:21:30 ON 15 DEC 2005

L1	15203 S MICROSPHER?
L2	343 S L1/TI
L3	990 S L1/AB
L4	1026 S L2 OR L3
L5	6164 S POLYANHYDRIDE
L6	2826 S SULINDAC
L7	16 S L6 AND L4
L8	3 S L7 AND L5
L9	16 S L7 AND POLYMER
L10	12 S L7 NOT PY>2002
L11	90719 S CANCER? OR TUMOR? OR CANCER?

L12 12 S L11 AND L10  
L13 89307 S POLYMERIC  
L14 89310 S L13 OR L9  
L15 189307 S POLYMER  
L16 208045 S L15 OR L13  
L17 16 S L16 AND L7  
L18 11 S L17 NOT PY>2001

=>

---Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSSPTA1642BJF

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

\*\*\*\*\* Welcome to STN International \*\*\*\*\*

NEWS 1 Web Page URLs for STN Seminar Schedule - N. America  
NEWS 2 "Ask CAS" for self-help around the clock  
NEWS 3 JAN 17 Pre-1988 INPI data added to MARPAT  
NEWS 4 FEB 21 STN AnaVist, Version 1.1, lets you share your STN AnaVist  
visualization results  
NEWS 5 FEB 22 The IPC thesaurus added to additional patent databases on STN  
NEWS 6 FEB 22 Updates in EPFULL; IPC 8 enhancements added  
NEWS 7 FEB 27 New STN AnaVist pricing effective March 1, 2006  
NEWS 8 MAR 03 Updates in PATDPA; addition of IPC 8 data without attributes  
NEWS 9 MAR 22 EMBASE is now updated on a daily basis  
NEWS 10 APR 03 New IPC 8 fields and IPC thesaurus added to PATDPAFULL  
NEWS 11 APR 03 Bibliographic data updates resume; new IPC 8 fields and IPC  
thesaurus added in PCTFULL  
NEWS 12 APR 04 STN AnaVist \$500 visualization usage credit offered  
NEWS 13 APR 12 LINSPEC, learning database for INSPEC, reloaded and enhanced  
NEWS 14 APR 12 Improved structure highlighting in FQHIT and QHIT display  
in MARPAT  
NEWS 15 APR 12 Derwent World Patents Index to be reloaded and enhanced during  
second quarter; strategies may be affected  
NEWS 16 MAY 10 CA/CAPLUS enhanced with 1900-1906 U.S. patent records  
NEWS 17 MAY 11 KOREAPAT updates resume  
NEWS 18 MAY 19 Derwent World Patents Index to be reloaded and enhanced

NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,  
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),  
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.  
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT  
<http://download.cas.org/express/v8.0-Discover/>

NEWS HOURS STN Operating Hours Plus Help Desk Availability  
NEWS LOGIN Welcome Banner and News Items  
NEWS IPC8 For general information regarding STN implementation of IPC 8  
NEWS X25 X.25 communication option no longer available after June 2006

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\*\*\*like to better understand what you find useful. Please take\*\*\*  
\*\*\*approximately 5 minutes to complete a web survey.\*\*\*

\*\*\*If you provide us with your name, login ID, and e-mail address, you\*\*\*  
\*\*\*will be entered in a drawing to win a free iPod(R). Your responses\*\*\*  
\*\*\*will be kept confidential and will help us make future improvements\*\*\*  
\*\*\*to STN.\*\*\*

\*\*\*Take survey: <http://www.zoomerang.com/survey.zgi?p=WEB2259HNKWTUW> \*\*\*

\*\*\*Thank you in advance for your participation.\*\*\*

\*\*\*\*\* STN Columbus \*\*\*\*\*

FILE 'HOME' ENTERED AT 14:54:10 ON 23 MAY 2006

=> file reg

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		0.21	0.21

FILE 'REGISTRY' ENTERED AT 14:54:22 ON 23 MAY 2006  
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Property values tagged with IC are from the ZIC/VINITI data file  
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STRUCTURE FILE UPDATES: 22 MAY 2006 HIGHEST RN 885262-53-3  
DICTIONARY FILE UPDATES: 22 MAY 2006 HIGHEST RN 885262-53-3

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

Please note that search-term pricing does apply when  
conducting SmartSELECT searches.

\*\*\*\*\*

\*  
\* The CA roles and document type information have been removed from \*  
\* the IDE default display format and the ED field has been added, \*  
\* effective March 20, 2005. A new display format, IDERL, is now \*  
\* available and contains the CA role and document type information. \*  
\*

\*\*\*\*\*

Structure search iteration limits have been increased. See HELP SLIMITS  
for details.

REGISTRY includes numerically searchable data for experimental and  
predicted properties as well as tags indicating availability of  
experimental property data in the original document. For information  
on property searching in REGISTRY, refer to:

<http://www.cas.org/ONLINE/UG/regprops.html>

=> E "SULINDAC"/CN 25

E1	1	SULIKOL K/CN
E2	1	SULIN/CN
E3	1 -->	SULINDAC/CN
E4	1	SULINDAC B .OMEGA.-N-METHYL-L-ARGININE SALT/CN

E5	1	SULINDAC B .OMEGA.-N-NITRO-L-ARGININE METHYL ESTER SALT/
E6	1	SULINDAC B .OMEGA.-N-NITRO-L-ARGININE SALT/CN
E7	1	SULINDAC ETHYL ESTER/CN
E8	1	SULINDAC SODIUM/CN
E9	1	SULINDAC SULFIDE/CN
E10	1	SULINDAC SULFONE/CN
E11	1	SULINDAC SULFOXIDE/CN
E12	1	SULINDAC-QUINOLINE/CN
E13	1	SULINEX/CN
E14	1	SULINOL/CN
E15	1	SULIODOVIZOL/CN
E16	1	SULISATIN/CN
E17	1	SULISATIN DISODIUM SALT/CN
E18	1	SULISATIN SODIUM/CN
E19	1	SULISATINE SODIUM/CN
E20	1	SULISOBENZONE/CN
E21	1	SULJEX/CN
E22	1	SULKA/CN
E23	1	SULKA K BOLUSES/CN
E24	1	SULKA N/CN
E25	1	SULKOR/CN

=> S E3

L1 1 SULINDAC/CN

=> DIS L1 1 SQIDE

THE ESTIMATED COST FOR THIS REQUEST IS 6.36 U.S. DOLLARS  
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2006 ACS on STN

RN 38194-50-2 REGISTRY

CN 1H-Indene-3-acetic acid, 5-fluoro-2-methyl-1-[[4-(methylsulfinyl)phenyl]methylene]-, (1Z)- (9CI) (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN 1H-Indene-3-acetic acid, 5-fluoro-2-methyl-1-[[4-(methylsulfinyl)phenyl]methylene]-, (Z)-

OTHER NAMES:

CN Aflodac

CN Algocetil

CN Arthrocline

CN Artribid

CN cis-5-Fluoro-2-methyl-1-[(p-methylsulfinyl)benzylidenyl]indene-3-acetic acid

CN cis-Sulindac

CN Citireuma

CN Clinoril

CN Clisundac

CN Imbaral

CN MK 231

CN Mobilin

CN Reumofil

CN Reumyl

CN Sudac

CN \*\*\*Sulindac\*\*\*

CN Sulindac sulfoxide

CN Sulinol

CN Sulreuma

FS STEREOSEARCH

DR 49627-22-7

MF C20 H17 F O3 S

CI COM

LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN\*, BIOSIS, BIOTECH CA, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDF DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IMSCOSEARCH, IMSPATENTS, IPA MEDLINE, MRCK\*, MSDS-OHS, PHAR, PROMT, PROUSDDR, PS, RTECS\*, SCI SPECINFO, TOXCENTER, USAN, USPAT2, USPATFULL, VETU

(\*File contains numerically searchable property data)

Other Sources: EINECS\*\*, WHO

(\*\*Enter CHEMLIST File for up-to-date regulatory information)

DT.CA Caplus document type: Conference; Dissertation; Journal; Patent

RL.P Roles from patents: ANST (Analytical study); BIOL (Biological study);

PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses)

RLD.P Roles for non-specific derivatives from patents: BIOL (Biological

study); PREP (Preparation); PROC (Process); RACT (Reactant or reagent);  
USES (Uses)  
RL.NP Roles from non-patents: ANST (Analytical study); BIOL (Biological  
study); FORM (Formation, nonpreparative); OCCU (Occurrence); PREP  
(Preparation); PROC (Process); PRP (Properties); RACT (Reactant or  
reagent); USES (Uses)  
RLD.NP Roles for non-specific derivatives from non-patents: ANST (Analytical  
study); BIOL (Biological study); PREP (Preparation); PRP (Properties);  
USES (Uses)

Double bond geometry as shown.

/ Structure 1 in file .gra /

**\*\*PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT\*\***

1474 REFERENCES IN FILE CA (1907 TO DATE)  
77 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA  
1477 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> file caplus

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		7.10	7.31

FILE 'CAPLUS' ENTERED AT 14:55:07 ON 23 MAY 2006  
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FILE COVERS 1907 - 23 May 2006 VOL 144 ISS 22  
FILE LAST UPDATED: 22 May 2006 (20060522/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.  
They are available for your review at:

<http://www.cas.org/infopolicy.html>

=> s l1  
L2 1477 L1

=> IL-12 or IL12 or (IL 12)  
IL-12 IS NOT A RECOGNIZED COMMAND  
The previous command name entered was not recognized by the system.  
For a list of commands available to you in the current file, enter  
"HELP COMMANDS" at an arrow prompt (=>).

=> s IL-12 or IL12 or (IL 12)  
114245 IL  
1077 ILS  
115018 IL  
(IL OR ILS)  
1386581 12  
9806 IL-12  
(IL(W)12)  
844 IL12  
114245 IL  
1077 ILS  
115018 IL

(IL OR ILS)  
1386581 12  
9806 IL 12  
(IL(W)12)  
L3 10023 IL-12 OR IL12 OR (IL 12)

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1477 L1  
L4 4 L3 AND L1

=> s I2 and I3  
L5 4 L2 AND L3

=> s I2 (L) I3  
L6 1 L2 (L) L3

=> d ibib

L6 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 2005:823135 CAPLUS  
DOCUMENT NUMBER: 143:210437  
TITLE: Autologous human dendritic cells fused with autologous  
cancer cells and human IL-12 for combined  
immunotherapy of cancer  
INVENTOR(S): Ohno, Tsuneya  
PATENT ASSIGNEE(S): USA  
SOURCE: U.S. Pat. Appl. Publ., 51 pp.  
CODEN: USXXCO  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005180951	A1	20050818	US 2004-778717	20040212
WO 2005079271	A2	20050901	WO 2005-US4237	20050211
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRIORITY APPLN. INFO.:			US 2004-778717	A 20040212

=> d I5 ibib 1-4

L5 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 2005:823135 CAPLUS  
DOCUMENT NUMBER: 143:210437  
TITLE: Autologous human dendritic cells fused with autologous  
cancer cells and human \*\*\*IL\*\*\* - \*\*\*12\*\*\* for  
combined immunotherapy of cancer  
INVENTOR(S): Ohno, Tsuneya  
PATENT ASSIGNEE(S): USA  
SOURCE: U.S. Pat. Appl. Publ., 51 pp.  
CODEN: USXXCO  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005180951	A1	20050818	US 2004-778717	20040212
WO 2005079271	A2	20050901	WO 2005-US4237	20050211
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,				



GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW  
 RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 2004-778717

A 20040212

L5 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:591975 CAPLUS

DOCUMENT NUMBER: 143:53482

TITLE: Method for inhibiting the growth of gastrointestinal tract tumors

INVENTOR(S): Egilmez, Nejat K.

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 21 pp.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005147689	A1	20050707	US 2003-748003	20031230
CA 2491338	AA	20050630	CA 2004-2491338	20041223

PRIORITY APPLN. INFO.: US 2003-748003 A 20031230

L5 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:570530 CAPLUS

DOCUMENT NUMBER: 143:91017

TITLE: Use of anti-inflammatory agent, anti-rheumatic agent, antihistamine and immunosuppressor in conjunction with liposome-mediated gene therapy to reduce inflammation

INVENTOR(S): Ramesh, Rajagopal; Gopalan, Began; Roth, Jack A.

PATENT ASSIGNEE(S): Board of Regents, the University of Texas System, USA

SOURCE: U.S. Pat. Appl. Publ., 61 pp.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005143336	A1	20050630	US 2004-341	20041130

PRIORITY APPLN. INFO.: US 2003-533180P P 20031230

L5 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:119752 CAPLUS

DOCUMENT NUMBER: 140:162347

TITLE: Compositions comprising tumor-dendritic Fusion cells, recombinant human interleukin 12, antipyretic and immunosuppressant for cancer immunotherapy

INVENTOR(S): Ohno, Tsuneya

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 38 pp., Cont.-in-part of U.S. Ser. No. 12,134.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004028663	A1	20040212	US 2002-328998	20021224
US 2002168351	A1	20021114	US 2001-12134	20011022

PRIORITY APPLN. INFO.: US 2000-242154P P 20001020  
 US 2001-12134 A2 20011022

=> s COX2 or (COX-2) or (COX 2)  
3949 COX2  
19709 COX  
2 COXES  
19711 COX  
(COX OR COXES)  
8741746 2  
10212 COX-2  
(COX(W)2)  
19709 COX  
2 COXES  
19711 COX  
(COX OR COXES)  
8741746 2  
10212 COX 2  
(COX(W)2)  
L7 11606 COX2 OR (COX-2) OR (COX 2)

=> d his

(FILE 'HOME' ENTERED AT 14:54:10 ON 23 MAY 2006)

FILE 'REGISTRY' ENTERED AT 14:54:22 ON 23 MAY 2006  
E "SULINDAC"/CN 25

L1 1 S E3

FILE 'CAPLUS' ENTERED AT 14:55:07 ON 23 MAY 2006

L2 1477 S L1  
L3 10023 S IL-12 OR IL12 OR (IL 12)  
L4 4 S L3 AND L1  
L5 4 S L2 AND L3  
L6 1 S L2 (L) L3  
L7 11606 S COX2 OR (COX-2) OR (COX 2)

=> s I7 and I3

L8 45 L7 AND L3

=> s I7 (L) I3

L9 39 L7 (L) L3

=> s synerg

=> s synerg?

L10 104630 SYNERG?

=> s I10 and I8

L11 0 L10 AND L8

=> s I10 and I9

L12 0 L10 AND L9

=> s I3 and I10

L13 645 L3 AND L10

=> s I13 and NSAID?

7454 NSAID?

L14 0 L13 AND NSAID?

=> file pctfull

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		32.78	40.09

FILE 'PCTFULL' ENTERED AT 15:00:05 ON 23 MAY 2006  
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FILE LAST UPDATED: 23 MAY 2006 <20060523/UP>  
MOST RECENT UPDATE WEEK: 200620 <200620/EW>  
FILE COVERS 1978 TO DATE

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>>> NEW IPC8 DATA AND FUNCTIONALITY NOW AVAILABLE IN THIS FILE.  
SEE

>>> FOR CHANGES IN PCTFULL PLEASE SEE HELP CHANGE  
(last updated April 10, 2006) <<<

=> s sulindac or clinoril or aflodac or mobilin

3129 SULINDAC

112 CLINORIL

1 CLINORILS

113 CLINORIL

(CLINORIL OR CLINORILS)

0 AFLODAC

1 MOBILIN

L15 3143 SULINDAC OR CLINORIL OR AFLODAC OR MOBILIN

=> s COX2 or (COX-2) or (COX 2)

500 COX2

10544 COX

1011013 2

4012 COX-2

(COX(W)2)

10544 COX

1011013 2

4012 COX 2

(COX(W)2)

L16 4331 COX2 OR (COX-2) OR (COX 2)

=> s synerg

L17 55 SYNERG

=> s synerg?

L18 35843 SYNERG?

=> s l16/ab

12 COX2/AB

374 COX/AB

207883 2/AB

296 COX-2/AB

((COX(W)2)/AB)

374 COX/AB

207883 2/AB

296 COX 2/AB

((COX(W)2)/AB)

L19 304 (COX2/AB OR (COX-2/AB) OR (COX 2/AB))

=> s l19 and l18

L20 65 L19 AND L18

=> s IL-12 or IL12 or (IL 12)

301684 IL

55063 ILS

320462 IL

(IL OR ILS)

911008 12

6676 IL-12

(IL(W)12)

582 IL12

301684 IL

55063 ILS

320462 IL

(IL OR ILS)

911008 12

6676 IL 12

(IL(W)12)

L21 6900 IL-12 OR IL12 OR (IL 12)

=> s l21 and l20

L22 7 L21 AND L20

=> s l21/clm

72231 IL/CLM

722730 12/CLM

992 IL-12/CLM

((IL(W)12)/CLM)

59 IL12/CLM  
72231 IL/CLM  
722730 12/CLM  
992 IL 12/CLM  
((IL(W)12)/CLM)  
L23 1033 (IL-12/CLM OR IL12/CLM OR (IL 12/CLM))

=> s l23 and l18  
L24 284 L23 AND L18

=> d his

(FILE 'HOME' ENTERED AT 14:54:10 ON 23 MAY 2006)

FILE 'REGISTRY' ENTERED AT 14:54:22 ON 23 MAY 2006  
E "SULINDAC"/CN 25

L1 1 S E3

FILE 'CAPLUS' ENTERED AT 14:55:07 ON 23 MAY 2006

L2 1477 S L1  
L3 10023 S IL-12 OR IL12 OR (IL 12)  
L4 4 S L3 AND L1  
L5 4 S L2 AND L3  
L6 1 S L2 (L) L3  
L7 11606 S COX2 OR (COX-2) OR (COX 2)  
L8 45 S L7 AND L3  
L9 39 S L7 (L) L3  
L10 104630 S SYNERG?  
L11 0 S L10 AND L8  
L12 0 S L10 AND L9  
L13 645 S L3 AND L10  
L14 0 S L13 AND NSAID?

FILE 'PCTFULL' ENTERED AT 15:00:05 ON 23 MAY 2006

L15 3143 S SULINDAC OR CLINORIL OR AFLODAC OR MOBILIN  
L16 4331 S COX2 OR (COX-2) OR (COX 2)  
L17 55 S SYNERG  
L18 35843 S SYNERG?  
L19 304 S L16/AB  
L20 65 S L19 AND L18  
L21 6900 S IL-12 OR IL12 OR (IL 12)  
L22 7 S L21 AND L20  
L23 1033 S L21/CLM  
L24 284 S L23 AND L18

=> s l24 and l15  
L25 22 L24 AND L15

=> s l15/clm  
490 SULINDAC/CLM  
9 CLINORIL/CLM  
0 AFLODAC/CLM  
0 MOBILIN/CLM  
L26 493 (SULINDAC/CLM OR CLINORIL/CLM OR AFLODAC/CLM OR MOBILIN/CLM)

=> s l26 and l25  
L27 13 L26 AND L25

=> s cancer? or tumor? or neoplas?  
78408 CANCER?  
65529 TUMOR?  
22767 NEOPLAS?  
L28 97667 CANCER? OR TUMOR? OR NEOPLAS?

=> s l27 and l28  
L29 12 L27 AND L28

=> s l18/clm  
L30 2678 (SYNERG?/CLM)

=> s l30 and l29  
L31 11 L30 AND L29

=> s l31 not py>2003

=> d ibib 1-5

L32 ANSWER 1 OF 10 PCTFULL COPYRIGHT 2006 Univentio on STN  
ACCESSION NUMBER: 2003030821 PCTFULL ED 20030428 EW 200316  
TITLE (ENGLISH): ALBUMIN FUSION FPROTEINS  
TITLE (FRENCH): PROTEINES DE FUSION D'ALBUMINE  
INVENTOR(S): ROSEN, Craig, A., 22400 Rolling Hill Lane,  
Laytonsville, MD 20882, US [US, US];  
HASELTINE, William, A., 3053 P Street, N.W.,  
Washington, DC 20007, US [US, US]  
PATENT ASSIGNEE(S): HUMAN GENOME SCIENCES, INC., 9410 Key West Aven  
Rockville, MD 20850, US [US, US], for all designates  
States except US;  
ROSEN, Craig, A., 22400 Rolling Hill Lane,  
Laytonsville, MD 20882, US [US, US], for US only;  
HASELTINE, William, A., 3053 P Street, N.W.,  
Washington, DC 20007, US [US, US], for US only  
AGENT: WALES, Michele, M.\$, Human Genome Sciences, Inc., 9410  
Key West Avenue, Rockville, MD 20850\$, US  
LANGUAGE OF FILING: English  
LANGUAGE OF PUBL.: English  
DOCUMENT TYPE: Patent  
PATENT INFORMATION:  
NUMBER KIND DATE  
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WO 2003030821 A2 20030417  
DESIGNATED STATES  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR  
CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID  
IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD  
MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI  
SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW  
RW (ARIPO): GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW  
RW (EAPO): AM AZ BY KG KZ MD RU TJ TM  
RW (EPO): AT BE BG CH CY CZ DE DK EE ES FI FR GB GR IE IT LU MC  
NL PT SE SK TR  
RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG  
APPLICATION INFO.: WO 2002-US31794 A 20021004  
PRIORITY INFO.: US 2001-60/327,281 20011005

L32 ANSWER 2 OF 10 PCTFULL COPYRIGHT 2006 Univentio on STN  
ACCESSION NUMBER: 2002077186 PCTFULL ED 20021011 EW 200240  
TITLE (ENGLISH): HUMAN SECRETED PROTEINS  
TITLE (FRENCH): PROTEINES SECRETEES PAR L'ETRE HUMAIN  
INVENTOR(S): ROSEN, Craig, A., 2240 Rolling Hill Lane, Laytonsville,  
MD 20882, US [US, US];  
RUBEN, Steven, M., 18528 Heritage Hills Drive, Olney,  
MD 20832, US [US, US]  
PATENT ASSIGNEE(S): HUMAN GENOME SCIENCES, INC., 9410 Key West Aven  
Rockville, MD 20850, US [US, US], for all designates  
States except US;  
ROSEN, Craig, A., 2240 Rolling Hill Lane, Laytonsville,  
MD 20882, US [US, US], for US only;  
RUBEN, Steven, M., 18528 Heritage Hills Drive, Olney,  
MD 20832, US [US, US], for US only  
AGENT: HOOVER, Kenley, K.\$, Human Genome Sciences, Inc., 9410  
Key West Avenue, Rockville, MD 20850\$, US  
LANGUAGE OF FILING: English  
LANGUAGE OF PUBL.: English  
DOCUMENT TYPE: Patent  
PATENT INFORMATION:  
NUMBER KIND DATE  
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WO 2002077186 A2 20021003  
DESIGNATED STATES  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR  
CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID  
IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD  
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TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW  
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RW (EPO): AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE  
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RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG  
APPLICATION INFO.: WO 2002-US9188 A 20020326  
PRIORITY INFO.: US 2001-60/278,650 20010327  
US 2001-09/950,082 20010912  
US 2001-09/950,083 20010912

L32 ANSWER 3 OF 10 PCTFULL COPYRIGHT 2006 Univentio on STN  
ACCESSION NUMBER: 2002072763 PCTFULL ED 20020927 EW 200238  
TITLE (ENGLISH): NUCLEIC ACIDS, PROTEINS, AND ANTIBODIES  
TITLE (FRENCH): ACIDES NUCLEIQUES, PROTEINES ET ANTICORPS  
INVENTOR(S): SHI, Yanggu, 437 West Side Drive, Apt. 102,  
Gaithersburg, MD 20878, US [US, US];  
NI, Jian, 17815 Fair Lady Way, Germantown, MD 20874, US  
[CN, US];  
RUBEN, Steven, M., 18528 Heritage Hills Drive, Olney,  
MD 20832, US [US, US]  
PATENT ASSIGNEE(S): HUMAN GENOME SCIENCES, INC., 9410 Key West Aven  
Rockville, MD 20850, US [US, US], for all designates  
States except US;  
SHI, Yanggu, 437 West Side Drive, Apt. 102,  
Gaithersburg, MD 20878, US [US, US], for US only;  
NI, Jian, 17815 Fair Lady Way, Germantown, MD 20874, US  
[CN, US], for US only;  
RUBEN, Steven, M., 18528 Heritage Hills Drive, Olney,  
MD 20832, US [US, US], for US only  
AGENT: HOOVER, Kenley, K.\$, Human Genome Sciences, Inc., 9410  
Key West Avenue, Rockville, MD 20850\$, US  
LANGUAGE OF FILING: English  
LANGUAGE OF PUBL.: English  
DOCUMENT TYPE: Patent  
PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 2002072763	A2	20020919
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DESIGNATED STATES

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR  
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MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI  
SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW

RW (ARIPO): GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW  
RW (EAPO): AM AZ BY KG KZ MD RU TJ TM  
RW (EPO): AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE  
TR

RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG  
APPLICATION INFO.: WO 2002-US6990 A 20020308  
PRIORITY INFO.: US 2001-60/274,214 20010309

L32 ANSWER 4 OF 10 PCTFULL COPYRIGHT 2006 Univentio on STN  
ACCESSION NUMBER: 2001055368 PCTFULL ED 20020827  
TITLE (ENGLISH): NUCLEIC ACIDS, PROTEINS, AND ANTIBODIES  
TITLE (FRENCH): ACIDES NUCLEIQUES, PROTEINES ET ANTICORPS  
INVENTOR(S): ROSEN, Craig, A.;  
BARASH, Steven, C.;  
RUBEN, Steven, M.  
PATENT ASSIGNEE(S): HUMAN GENOME SCIENCES, INC.;  
ROSEN, Craig, A.;  
BARASH, Steven, C.;  
RUBEN, Steven, M.

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 2001055368	A1	20010802
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DESIGNATED STATES

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU  
CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN  
IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK  
MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM  
TR TT TZ UA UG US UZ VN YU ZA ZW GH GM KE LS MW MZ SD  
SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY

DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF  
CG CI CM GA GN GW ML MR NE SN TD TG  
APPLICATION INFO.: WO 2001-US1348 A 20010117  
PRIORITY INFO.: US 2000-60/179,065 20000131

US 2000-60/180,628	20000204
US 2000-60/184,664	20000224
US 2000-60/186,350	20000302
US 2000-60/189,874	20000316
US 2000-60/190,076	20000317
US 2000-60/198,123	20000418
US 2000-60/205,515	20000519
US 2000-60/209,467	20000607
US 2000-60/214,886	20000628
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US 2000-60/218,290	20000714
US 2000-60/220,963	20000726
US 2000-60/220,964	20000726
US 2000-60/225,757	20000814
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US 2000-60/254,097	20001211
US 2001-60/259,678	20010105

L32 ANSWER 5 OF 10 PCTFULL COPYRIGHT 2006 Univentio on STN  
 ACCESSION NUMBER: 2001055328 PCTFULL ED 20020827  
 TITLE (ENGLISH): NUCLEIC ACIDS, PROTEINS, AND ANTIBODIES  
 TITLE (FRENCH): ACIDES NUCLEIQUES, PROTEINES ET ANTICORPS  
 INVENTOR(S): ROSEN, Craig, A.;  
                   BARASH, Steven, C.;  
                   RUBEN, Steven, M.  
 PATENT ASSIGNEE(S): HUMAN GENOME SCIENCES, INC.;



ROSEN, Craig, A.;  
BARASH, Steven, C.;  
RUBEN, Steven, M.

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER KIND DATE

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WO 2001055328 A2 20010802

DESIGNATED STATES

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SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY  
DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF  
CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2001-US1359 A 20010117

PRIORITY INFO.: US 2000-60/179,065 20000131

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US 2000-60/251,990	20001208
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US 2000-60/254,097	20001211
US 2001-60/259,678	20010105

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L32 ANSWER 6 OF 10 PCTFULL COPYRIGHT 2006 Univentio on STN  
 ACCESSION NUMBER: 2001055205 PCTFULL ED 20020827  
 TITLE (ENGLISH): NUCLEIC ACIDS, PROTEINS, AND ANTIBODIES  
 TITLE (FRENCH): ACIDES NUCLEIQUES, PROTEINES ET ANTICORPS  
 INVENTOR(S): ROSEN, Craig, A.;  
 BARASH, Steven, C.;  
 RUBEN, Steven, M.  
 PATENT ASSIGNEE(S): HUMAN GENOME SCIENCES, INC.;  
 ROSEN, Craig, A.;  
 BARASH, Steven, C.;  
 RUBEN, Steven, M.

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 2001055205	A1	20010802
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DESIGNATED STATES

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 IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK  
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 SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY  
 DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF  
 CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2001-US1337 A 20010117

PRIORITY INFO.: US 2000-60/179,065 20000131

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US 2000-60/251,990	20001208
US 2000-60/251,989	20001208
US 2000-60/254,097	20001211
US 2001-60/259,678	20010105

L32 ANSWER 7 OF 10 PCTFULL COPYRIGHT 2006 Univentio on STN  
 ACCESSION NUMBER: 2001055203 PCTFULL ED 20020827  
 TITLE (ENGLISH): NUCLEIC ACIDS, PROTEINS, AND ANTIBODIES  
 TITLE (FRENCH): ACIDES NUCLEIQUES, PROTEINES ET ANTICORPS  
 INVENTOR(S): ROSEN, Craig, A.;

BARASH, Steven, C.;

RUBEN, Steven, M.

PATENT ASSIGNEE(S): HUMAN GENOME SCIENCES, INC.;

ROSEN, Craig, A.;

BARASH, Steven, C.;

RUBEN, Steven, M.

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 2001055203	A1	20010802
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DESIGNATED STATES

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 TR TT TZ UA UG US UZ VN YU ZA ZW GH GM KE LS MW MZ SD  
 SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY  
 DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF  
 CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2001-US1327 A 20010117

PRIORITY INFO.: US 2000-60/179,065 20000131

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US 2000-60/251,868	20001208
US 2000-60/251,990	20001208
US 2000-60/251,989	20001208
US 2000-60/254,097	20001211
US 2001-60/259,678	20010105

L32 ANSWER 8 OF 10 PCTFULL COPYRIGHT 2006 Univentio on STN  
 ACCESSION NUMBER: 2001055201 PCTFULL ED 20020827  
 TITLE (ENGLISH): NUCLEIC ACIDS, PROTEINS, AND ANTIBODIES  
 TITLE (FRENCH): ACIDES NUCLEIQUES, PROTEINES ET ANTICORPS  
 INVENTOR(S): ROSEN, Craig, A.;

BARASH, Steven, C.;

RUBEN, Steven, M.

PATENT ASSIGNEE(S): HUMAN GENOME SCIENCES, INC.;

ROSEN, Craig, A.;

BARASH, Steven, C.;

RUBEN, Steven, M.

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 2001055201	A1	20010802
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DESIGNATED STATES

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU  
 CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN  
 IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK  
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 CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2001-US1317 A 20010117

PRIORITY INFO.: US 2000-60/179,065 20000131

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US 2000-60/236,802	20001002
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US 2000-60/239,935	20001013
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US 2000-60/251,989	20001208
US 2000-60/254,097	20001211
US 2001-60/259,678	20010105

L32 ANSWER 9 OF 10 PCTFULL COPYRIGHT 2006 Univentio on STN  
 ACCESSION NUMBER: 2001055164 PCTFULL ED 20020827  
 TITLE (ENGLISH): NUCLEIC ACIDS, PROTEINS, AND ANTIBODIES  
 TITLE (FRENCH): ACIDES NUCLEIQUES, PROTEINES ET ANTICORPS  
 INVENTOR(S): ROSEN, Craig, A.;  
 BARASH, Steven, C.;  
 RUBEN, Steven, M.  
 PATENT ASSIGNEE(S): HUMAN GENOME SCIENCES, INC.;  
 ROSEN, Craig, A.;  
 BARASH, Steven, C.;  
 RUBEN, Steven, M.

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 2001055164	A1	20010802
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DESIGNATED STATES

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU  
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TR TT TZ UA UG US UZ VN YU ZA ZW GH GM KE LS MW MZ SD  
SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY  
DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF  
CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2001-US1566 A 20010117  
PRIORITY INFO.: US 2000-60/179,065 20000131

US 2000-60/180,628	20000204
US 2000-60/184,664	20000224
US 2000-60/189,874	20000316
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US 2000-60/251,479	20001206
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L32 ANSWER 10 OF 10 PCTFULL COPYRIGHT 2006 Univentio on STN  
ACCESSION NUMBER: 2001054733 PCTFULL ED 20020827

TITLE (ENGLISH): NUCLEIC ACIDS, PROTEINS AND ANTIBODIES

TITLE (FRENCH): ACIDES NUCLEIQUES, PROTEINES ET ANTICORPS

INVENTOR(S): ROSEN, Craig, A.;

BARASH, Steven, C.;

RUBEN, Steven, M.

PATENT ASSIGNEE(S): HUMAN GENOME SCIENCES, INC.;

ROSEN, Craig, A.;

BARASH, Steven, C.;

RUBEN, Steven, M.

DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
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WO 2001054733	A1	20010802
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DESIGNATED STATES

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CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2001-US1312 A 20010117  
PRIORITY INFO.: US 2000-60/179,065 20000131

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